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THE BLOOD VESSELS OF OSTEOGENIC SARCOMAS

Histologic, angiographic and microradiographic studies

by

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It has long been known that some osteogenic sarcomas are highly vascular. The skin over such tumors is warmer than that covering the surrounding tissues, and the subcutaneous veins are dilated. CODMAN has called this type of tumor a teleangiectatic osteogenic sarcoma. The high vascularity has been repeatedly demonstrated by means of angiography, and when examined by this method most osteogenic sarcomas are found to be considerably richer in vessels than the surrounding normal tissues (BEGG, COLUMELLA & MUCCHI, FARIÑAS, FONTAINE et coll., STRICKLAND, SUTTON, TIWISINA, VOGLER & DEU). We have found, however, that some osteogenic sarcomas have only a slightly increased vascularity. VOGLER & DEU's material also included three 'bone sarcomas' in which the number of vessels was not abnormally high. Since in recent years angiography has come to be used to an increasing extent as an aid in the diagnosis of bone tumors, it was considered that a closer study of the vessels in these tumors might explain their variations in vascularity.

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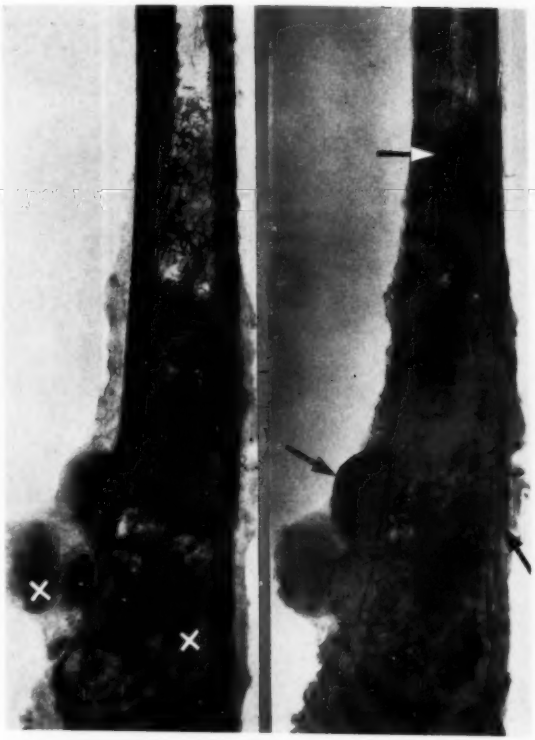
Material. Twenty-four cases of histologically confirmed osteogenic sarcomas were examined; angiography was performed in 21, none of which had previously received radiation therapy. In 13 of these cases, and in 3 others that had not been submitted to angiography, arteriography was performed on the amputation specimen. The vessels were examined both by combined microangiographic and histologic techniques described in an earlier report on the vessels of fibrosarcomas (LAGERGREN et coll.). A selection was made of parts of the tumor with different vascular structures as seen in the roentgenograms of decalcified sections, 0.5 cm thick, through the tumor. The degree of mineralization and the structure of the bony tissue of the tumor were studied by a microradiographic technique (ENGSTRÖM), representative areas being taken from undecalcified sections that had been embedded in methylmethacrylate and ground to a thickness of 100 to 200 microns. ENGFELDT, in his studies of the microradiographic structure of osteogenic sarcoma observed a wide variation in the appearance and degree of mineralization of the tumor bone.

Histologic findings

The tumors were classified according to the systems of JAFFE and DAHLIN. Three were labelled as juxtacortical osteogenic sarcoma, 12 as osteoblastic, 4 as chondroblastic and 5 as fibroblastic osteogenic sarcoma.

The vessels were readily identified in the histologic sections of the specimens containing contrast medium. There was a wide variation in the degree of bone formation in some of the osteoblastic sarcomas, some parts being as hard as cortical bone while others were not so firm and occasionally quite soft. Roentgenograms of a 0.5 cm thick section through such a tumor before and after decalcification are reproduced in Fig. 1. There was a soft, central extension of the growth into the medullary cavity of the diaphysis with a high cell density and little osteoid tissue and a low degree of differentiation to mineralized tumor bone. Many blood vessels in this part of the tumor had thin walls, resembling those of capillaries, with a single layer of endothelial cells or fibroblasts. The vessels had a pathologic irregular ramification and a wide variation in calibre (maximum 300 microns); some were sac-like in form (Fig. 2, a and b); no elastic fibres were detected. This tumor structure in certain sections extended into the cortical layer, but it alternated with areas of moderate formation of osteoid tissue and a differentiation to irregular tumor bone. These areas contained fewer cells which were less irregular and presented fewer mitotic figures than the undifferentiated areas described above. The vessels were as a rule less abundant and more narrow (maximum calibre about 150 microns) and were seen to course in the spaces between the trabeculae of osteoid tissue and bone (Fig. 2, c and d). These vessels also contained no elastic fibres. A part of the tumor, of smooth contour and very hard consistency, protruded outside the corti-



a b

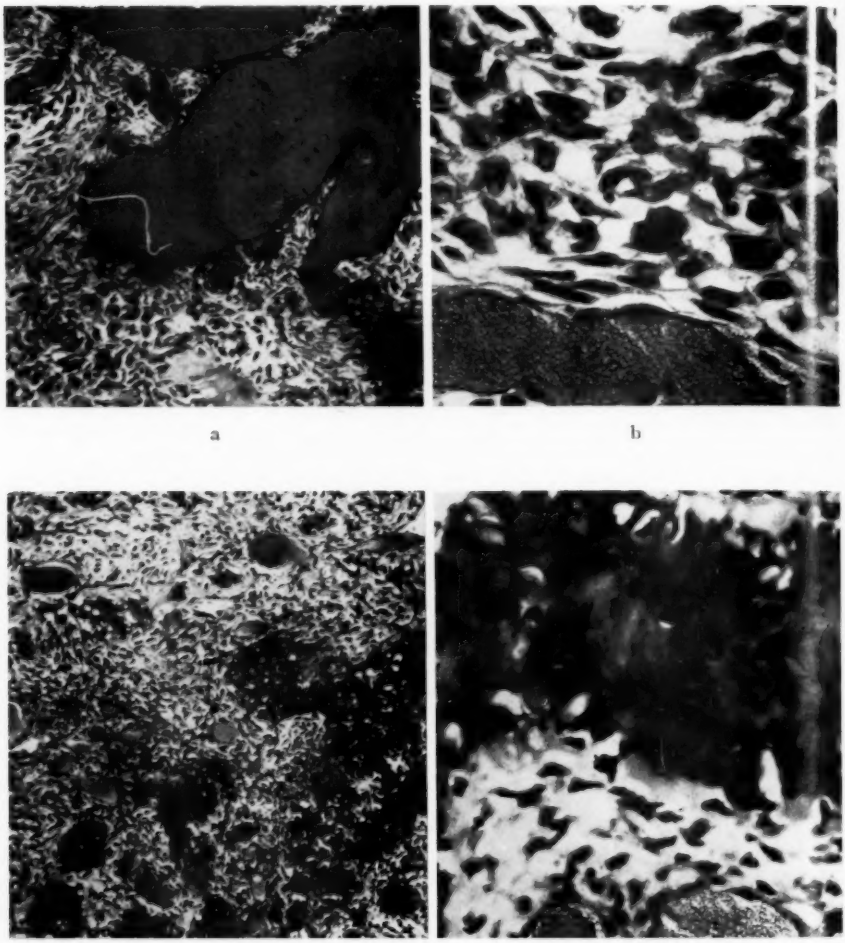
Fig. 1. Roentgenograms of a 0.5 cm section of injected osteoblastic osteogenic sarcoma in distal metaphysis of the femur.

a) Ordinary bone and bone-forming tumor tissue intensively black. Tumor occupies the medullary cavity and protrudes with bony parts posteriorly outside cortex. Microradiograms (see fig. 3, c and d) were taken from adjacent sections, in areas with same structure as those indicated by a cross.

b) After decalcification. Injected vessels are black, indicating extension of hypervascularized tumor. Heavily calcified parts are less vascular than the soft parts of the tumor in the medullary cavity, cf. (a). Photomicrographs (see fig. 2) and microangiograms (see fig. 8) were taken from the areas indicated by arrows.

cal bone (Fig. 1) and contained a dense mesh of osteoid trabeculae with differentiation of mineralized tumor bone (Fig. 2, e and f). The mineralization of the neoplastic bone was irregular. The tumor bone trabeculae in some regions ran perpendicular to the cortical bone and in the relatively narrow spaces between the trabeculae small blood vessels of the capillary type (maximum calibre about 80 microns) were noted (Fig. 2 e). There was no appreciable differentiation of the vessel walls in the tumor tissue, but elastic fibres were observed in a few of the vessels. The mineralization of the tumor bone was clearly defined with Heidenhain's staining method, which is usually employed to demonstrate the striation of muscles; the striations, erythrocytes and mineralized bone appeared coal-black, while the osteoid tissue, with incomplete mineralization, assumed different shades of grey (Fig. 3, a and b).

Regions with as great a range of differentiation as found in this tumor were

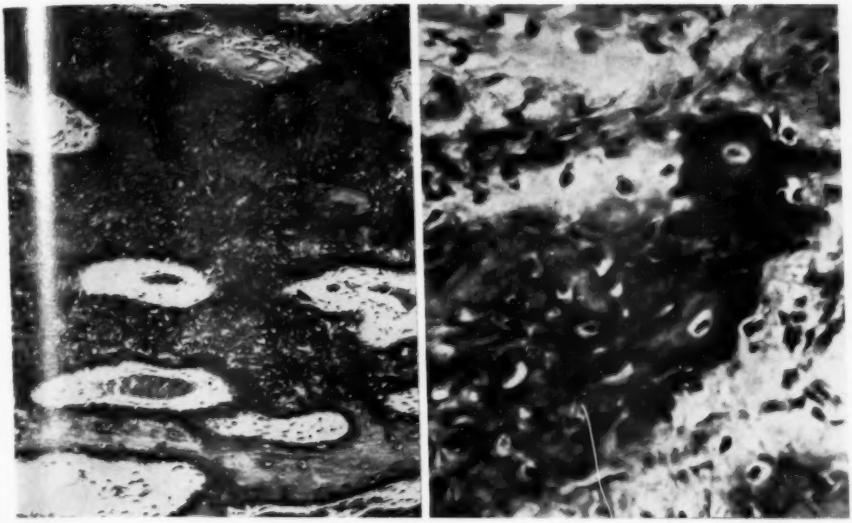


a b

c d

Fig. 2. Photomicrographs of three different regions of the same osteogenic sarcoma as shown in fig. 1. a) × 85. Poorly differentiated tumor extending into medullary cavity of diaphysis (upper arrow in fig. 1 b). Dilated blood vessels containing contrast medium. b) × 350. Higher magnification of (a). Anaplastic cells in non-ossified part of tumor. c) × 85. Intra-osseous part (lower right arrow in fig. 1b) with calcified osteoid and slightly dilated blood vessels. d) × 350. Higher magnification of (c). Moderately atypical osteoblasts and osteoid.

(For Fig. 2, e and f, see opposite page.)



e f

Fig. 2. e) × 85. Sclerotic, highly differentiated part of tumor outside the cortex (lower left arrow in fig. 1b) with capillary blood vessels between the bone trabeculae. f) × 350. High magnification of (e). Calcified osteoid and tumor osteoblasts with slightly to moderately atypical nuclei.

(For Fig. 2, a to d, see opposite page.)

distinguished also in several other tumors, but as a rule the difference between the areas was not so marked. It was observed in several instances that vessels of a normal appearance penetrated a few millimetres into the peripheral part of the tumor tissue, but further inwards they were destroyed and replaced by abnormal vessels.

The same primary features were found in a juxtacortical osteogenic sarcoma of the distal metaphysis of the femur (Fig. 4). The outermost layer in the parosteal region was surrounded by a zone of connective tissue that constituted a boundary between the tumor and the soft tissues. A narrow zone of spindle cells with a moderate degree of nuclear and cellular polymorphism and fine streaks of osteoid tissue were evident beneath this layer. There was more highly differentiated osteoid tissue and mineralized tumor bone, organized into a dense trabecular framework, below this in the major part of the parosteal region; tumor tissue, which was more cellular than polymorphous lay in the spaces of this framework. The blood vessels were thin-walled with occasional elastic fibres, but with no distinct differentiation into arteries and veins; they were of fairly small calibre and coursed in the spaces

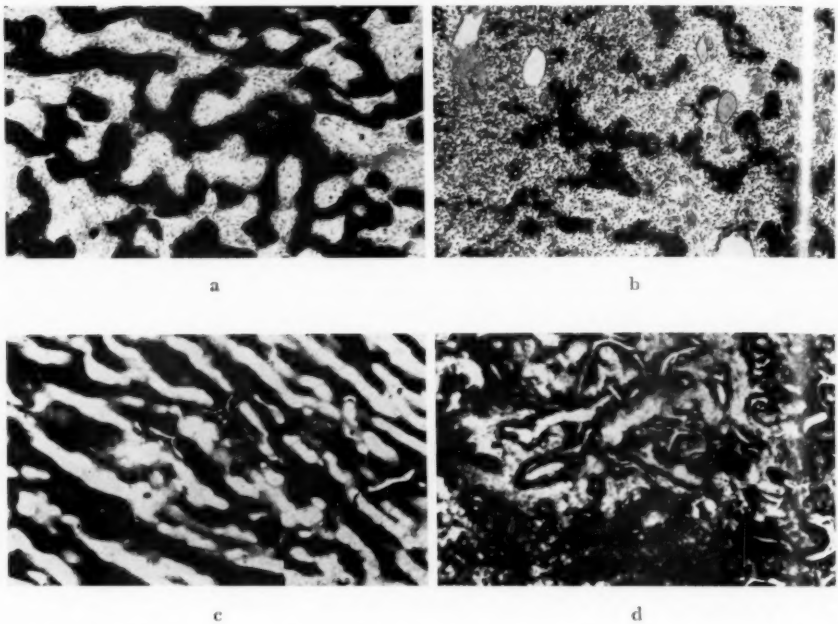


Fig. 3. Differentiation of neoplastic bone shown histologically and microradiographically. a) and b) Photomicrographs, Heidenhain's stain, × 35. Neoplastic bone trabeculae (black). a) Well differentiated part of tumor. b) Less differentiated part, trabeculae less mineralized, more irregular and sparse. c) and d) Microradiograms of approx. 100 micron sections × 35. Bone trabeculae (white), the reverse of (a) and (b). Sharply defined white structures are vessels filled with contrast medium. c) Same part as in (a). Abnormal bone trabeculae, well differentiated. d) Same part as in (b). Abnormal bone trabeculae, poorly differentiated.

between the trabeculae of osteoid tissue and bone. An intermediate zone, which included the cortex and the subcortical region was present in this tumor; this contained a large quantity of osteoid tissue in the trabeculae, but the mineralization was considerably less marked here than in the parosteal part. Centrally in the metaphysis lay a poorly differentiated zone, probably proliferating as a secondary process into the spongy bone of the metaphysis, and composed of highly cellular tumor tissue with practically no osteoid tissue and only a few mineralized patches. The vessels in this zone were irregular, thin-walled and wide (maximum calibre 300 microns) and much more numerous than elsewhere in the tumor. The cells were polymorphous predominantly spindle-shaped, with some of them stellate. Small areas of necrosis were also found in this zone.

a b

Fig. 4. Juxtacortical osteogenic sarcoma in typical location on popliteal surface of femur. a) Arteriogram in vivo. Slight opacity of region in medullary cavity (arrows). b) Decalcified 0.5 cm section of injected specimen (remnants of calcium salt in cortical layers). Increased vascularity causes tumor to stand out darker against surrounding bone. Part of tumor, growing into medullary cavity, more vascular than the rest of it. Cf. microangiograms and microradiograms in fig. 9.

Lobules of cartilaginous tissue with highly irregular cells were observed in two chondroblastic osteogenic sarcomas situated proximally in the tibia. The blood vessels were situated in curved stretches of interlobular connective tissue. Similar structures are found in a chondrosarcoma. Areas of less differentiated sarcoma tissue, with spindle or polyhedral cells having irregular chromatin-rich nuclei with islands of osteoid tissue and mineralized tumor bone and occasional irregular multinuclear giant cells, were present in addition to the chondromatous lobuli. The blood vessels in the connective tissue

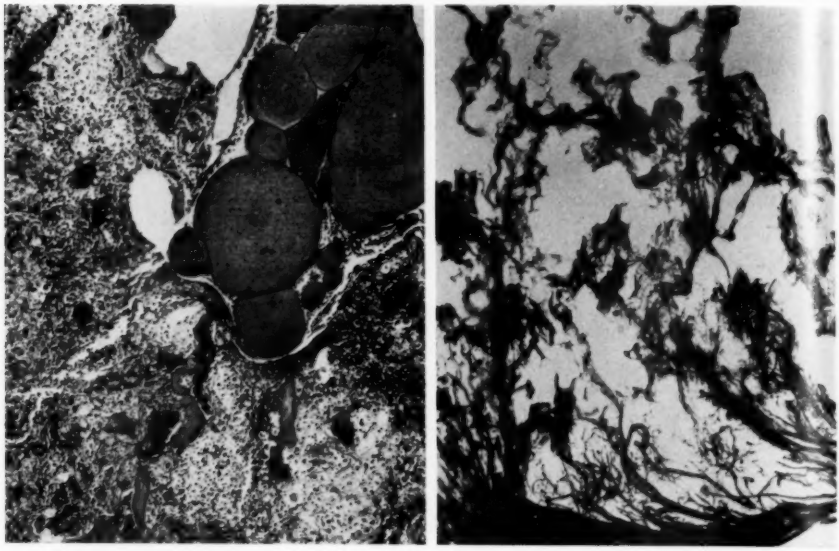


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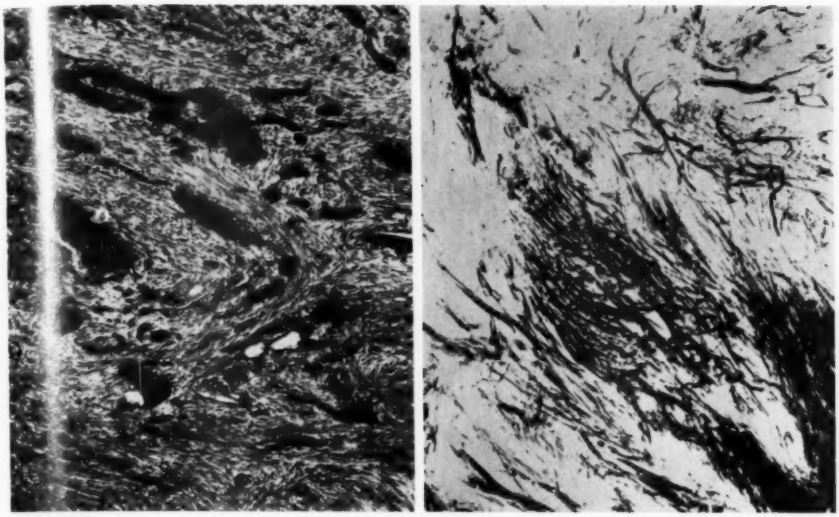
Fig. 5. Vascular structure of chondroblastic osteogenic sarcoma. a) Photomicrograph × 35. Large vessels, filled with contrast medium, between lobules of chondromatous neoplastic tissue. b) Microangiogram × 12. Lobular pattern of condrosarcoma. Wide, curved interlobular vessels.

between the lobuli were often very wide and thin-walled (maximum calibre 500 microns) (Fig. 5 a). The blood vessels in the cell-rich and less differentiated parts of the tumor were more narrow and less regularly arranged and did not follow the curved course that was observed in the more highly differentiated cartilaginous parts.

One fibroblastic osteogenic sarcoma, situated in the proximal metaphysis of the tibia, had a low degree of differentiation and little osteoid tissue. This tumor contained highly irregular spindle cells with atypical chromatin-rich nuclei and small amounts of fibrillar stroma. The vessels were in parts very wide (maximum calibre 500 microns); the vessel walls were extremely thin, and the impression was gained that the tumor cells in some places actually constituted the wall of the vessels. The vessels coursed circuitously but in a general direction, with convolutions and small irregular, sac-like and bud-like formations; they were densely spaced and in the main followed the general direction of the spindle cells of the tumor (Fig. 6 a). Another fibroblastic osteogenic sarcoma of the distal metaphysis of the femur had similar features.

Prior to the injection of the contrast medium, the amputation specimen



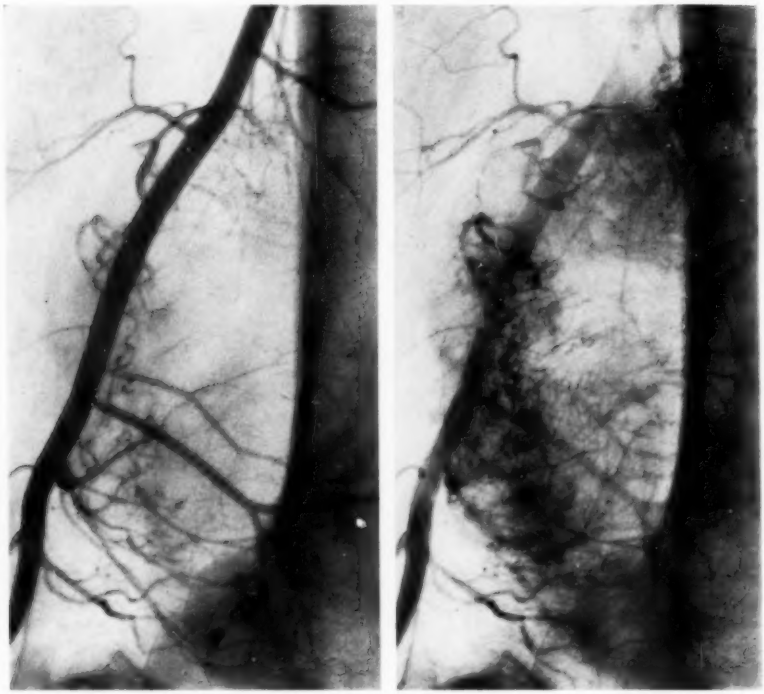
a b

Fig. 6. Vascular structure of fibroblastic osteogenic sarcoma. a) Photomicrograph × 35. Mainly fibrosarcomatous tissue with many drawn-out vessels, filled with contrast medium, arranged in the same general direction as the fibroblasts. b) Microangiogram × 9. Streaky pattern, sometimes also seen in well differentiated fibrosarcomas.

was in a few cases perfused with normal saline containing glass spheres of various sizes (PRINZMETAL et coll.). In one case of fibroblastic osteogenic sarcoma the venous efflux was found to contain glass spheres, most of them up to 50 microns, but some as large as 70 microns in diameter. In the histologic sections many of the pearls were found caught in the tumor vessels, and from this it may be inferred that there were shunts between the arteries and veins through the tumor; these vessels had a diameter of 70 microns.

Angiographic findings

Serial films were obtained in practically all the in vivo angiographic examinations. An increase in the number of arteries around the tumor, the presence of pathologic vessels within it (Fig. 7), and an increase in the veins leaving the tumor area, were noted. The fact that these veins were filled with contrast medium earlier in the course of angiography than other veins in the surrounding tissues was regarded as evidence of shunting through the tumor. The degree of vascularity of the tumors was estimated on the basis

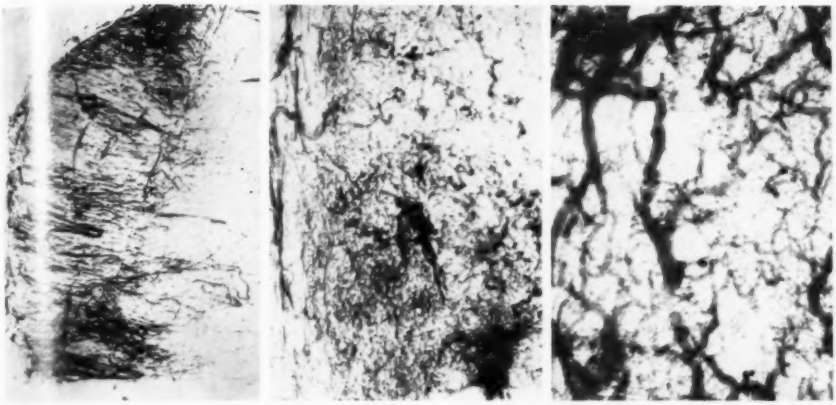


a b

Fig. 7. Pathologic tumor vessels in extra-osseous part of osteoblastic osteogenic sarcoma in distal end of femur. Serial angiography in vivo. In early arterial phase (a) a few vessels filled with contrast medium; two seconds later (b) typical dense network of pathologic vessels.

of these findings. In all cases the vascularity was greater in the tumor than in the surrounding tissues and ranged from moderate to marked.

The extent to which the bone was destroyed or new bone was formed in each tumor was estimated in ordinary roentgenograms; the vascularity of the tumors was then compared with the degree of destruction and sclerosis. The 13 cases presenting a high degree of vascularity all had destruction of bone, and in most of the tumors this tendency was more marked than the sclerosis; this was especially true in the more highly vascular cases. The 8 cases in which the vascularity was moderate showed a more or less marked formation of bone, and this tendency was distinctly more evident than bone destruction. Three tumors with no appreciable bone destruction had only slightly or moderately greater vascularity than the surrounding tissues.



a b c

Fig. 8. Vascularity in three different parts of same osteoblastic osteogenic sarcoma. Microangiograms × 9 of regions seen in figs 1 and 2. a) Well differentiated, bony part. Narrow, parallel vessels. b) Intermediate zone. Wider, more irregular vessels. c) Poorly differentiated zone, soft part. Very wide irregular vessels.

In 12 of the 21 cases there was a highly vascular soft part of the tumor outside the bone. In several cases this contained thin streaks of tumor bone, which were visible in the ordinary roentgenogram, although only in the angiogram was it possible to judge the size of this part of the tumor; in some cases this part of the tumor did not contain bone and was visible only in the angiogram. In 4 cases a highly vascular part of the tumor lay in the medullary cavity. The boundary of the tumor in the latter could then be judged in the angiograms, and in several cases it was found to have grown considerably more than was evident from the ordinary roentgenogram, as the cortical bone corresponding to this highly vascular part of the tumor was intact (See Fig. 1).

The vascularity of the tumor in several cases in which the bone had not been penetrated was difficult to judge since in spite of satisfactory filling with contrast medium the vessels could not be distinguished through the often sclerosed bone. The vessels were more easily demonstrated when higher kilovoltage and more concentrated (60 %) sodium diatrizoate was used.

Microradiographic findings

It was evident from the microangiograms that all the tumors examined were at least in parts more highly vascular than the surrounding tissues. Several of the osteoblastic tumors were markedly heterogeneous, some regions

containing very dense wide vessels in an irregular reticular formation and others sparser, narrower, regular, and often parallel vessels (See Fig. 1). The highly vascular areas corresponded in these tumors to the less differentiated areas where there was less new bone formation, while the sparser vessels were found in parts where there was considerable new bone (Fig. 8). In one case of juxtacortical osteogenic sarcoma in the femur with an apparently secondary growth into the medullary cavity, the same difference between the various parts of the tumor was evident. The part of the tumor outside the femur in which the bone-forming process was marked, thus contained more vessels than the normal cortical layer; they were, however, more regular and often more parallel than in the more highly and irregularly vascular and less differentiated parts of the tumor growing in the cavities of the spongy bone in the metaphysis (Fig. 9, a and c). The vessels in the chondroblastic tumors followed a characteristic curved course between the cartilaginous lobules in the connective tissue and were often extremely wide and flattened (Fig. 5 b). The microangiograms in the fibroblastic tumors showed bundles of wide parallel vessels winding around one another (Fig. 6 b). These growths thus exhibited a different vascular structure from the osteoblastic tumors.

The microradiographic examinations showed that the bony tissue of the tumors varied widely in its appearance and degree of mineralization, and that even when it was highly differentiated, the new bone differed from normal bone. There were extremely few ordinary Haversian systems, and only traces of the regular lamellar structure of normal bone were found. On the other hand, the fragments of residual normal bone which had not been destroyed by the tumor tissue presented the characteristic organization, with young, little mineralized and old, highly mineralized, Haversian systems.

The highly differentiated parts of the sarcoma had a very regular appearance, with smooth uniform trabeculae with a high mineral content (Fig. 9 b); in the sclerosed regions the trabeculae were often very densely arranged, more so than in normal spongy bone (Fig. 9 b). The bone formed symmetric, long, parallel trabeculae in some of the highly differentiated areas (Fig. 3 c).

The appearances were less uniform, with only a few large irregular trabeculae in the regions with a low degree of differentiation. These trabeculae had a fairly high degree of mineralization and were surrounded by poorly, or sometimes only partly, mineralized trabeculae of varying thickness and direction (Fig. 3 d). Where the degree of differentiation was still lower there was no evidence of trabeculae but irregularly dispersed patches with a varied but always low content of mineral salts were present; these patches sometimes merged to form a mossy network (Fig. 9 d).

In some of the cases with an extra-osseous tumor growth but with no tumor bone visible in the ordinary roentgenogram the microradiograms showed small mineralized regions with a varying degree of differentiation.

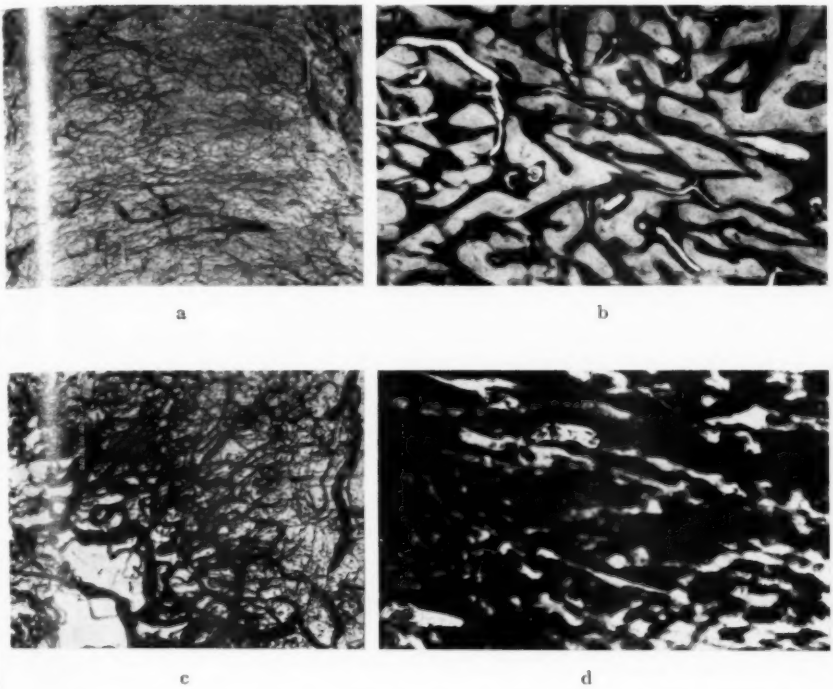


Fig. 9. Vascular and microradiographic structures of the juxta-cortical osteogenic sarcoma shown in fig. 4. a) Microangiogram × 9. Well differentiated part. b) Microradiogram × 35 of region similar to the one in (a). Coarse white structures are bone trabeculae and fine white streaks are vessels. Abnormal trabeculae dense and regular. c) Microangiogram × 9. Poorly differentiated part. d) Microradiogram × 35 of region similar to the one in (c). Abnormal trabeculae narrow and poorly mineralized.

Discussion

Most osteoblastic osteogenic sarcomas are very heterogenous tumors, that is to say, some parts of the tumor are highly differentiated with considerable new bone formation, whereas other parts are poorly differentiated with the formation only of osteoid tissue and little, if any, bone. In the parts of the tumor where the microradiograms showed a sparse, poorly organized formation of bone, the tumor tissue had a histologically more malignant appearance, with marked cellular irregularity and a high frequency of mitotic figures. Heidenhain staining revealed black structures very similar to the white structures in the microradiograms. There was a close agreement between the microradiographic and histologic structures of the different parts of the tumor

(Fig. 3). The vessels in the poorly differentiated regions were extremely irregular and wide (cf. Fig. 2, a and b with Fig. 8 c). In the regions where on the other hand, fairly regular trabeculae were observed in the microradiograms and where the tumor tissue was histologically less malignant, the vessels were narrower and more regular (cf. Fig. 2, e and f with Fig. 8 a); a few elastic fibres could be seen in the vessel walls, which suggested some degree of differentiation also of the vessels. In an earlier paper on fibrosarcomas we have presented evidence of a relationship between the vascularity and the degree of malignancy in these tumors. The same conclusion seems to apply to the osteogenic sarcomas. The less differentiated parts of these tumors with little new bone formation are much more vascular than the more highly differentiated parts where there is massive bone formation and where the vascularity is not much greater than in the normal tissue.

On angiography the juxtacortical osteogenic sarcoma with considerable new bone formation presented only a moderately increased vascularity in relation to the surrounding tissues. These highly differentiated tumors have probably as a rule no poorly differentiated malignant and vascular components, which possibly explains their considerably more benign nature compared to the ordinary osteogenic sarcoma. JAFFE distinguishes two types of juxtacortical osteogenic sarcoma, one of which has a more malignant course. When angiography reveals a highly vascular region in such a tumor, it is probably of the more malignant type (see Fig. 4).

Angiography in most of the ordinary heterogeneous osteoblastic osteogenic sarcomas revealed highly vascular parts from which cells or cell aggregates might more easily be carried away to the lungs than from the less richly vascular parts. The angiographic demonstration of these highly vascular parts of the tumor constitutes a valuable support for the diagnosis.

A histologic diagnosis is generally desirable in these cases, especially when amputation is intended. A soft tumor component growing out from the bone can often be demonstrated angiographically, and in such cases it is best to take a biopsy specimen from this region in order to obtain as early a diagnosis as possible (Fig. 7). In some cases the angiograms will indicate how far the tumor has grown into the medullary cavity. A difference in the microangiographic structure of the various types of osteogenic sarcoma can be demonstrated. The chondroblastic parts of the tumor have a lobular structure which has commonly been observed in the chondrosarcoma; the fibroblastic tumors have a streaky structure, which has been observed in some previous cases of fibrosarcomas. It is thus possible, in some cases at least, to recognize the various tumor tissues by their microangiographic structure (cf. Figs 5 b, 6 b and 8). In the ordinary in vivo angiographic examinations, however, only the coarser vascular structures will be visible so that it is uncertain whether the various types of tumor can be distinguished with the present angiographic techniques.

The following conclusions may be drawn with respect to the angiographic diagnosis:

The juxtacortical osteogenic sarcoma is only slightly more vascular than the surrounding tissue. With a secondary growth into the marrow space the tumor may be highly vascular.

An osteoblastic osteogenic sarcoma usually contains highly vascular parts which can be demonstrated in the angiogram; this feature provides a valuable confirmation of the diagnosis.

A few osteoblastic osteogenic sarcomas which display little tendency to osteolysis are only slightly more vascular than the surrounding tissue.

It is probable that fibroblastic and chondroblastic osteogenic sarcomas are as a rule highly vascular.

SUMMARY

Twenty-four cases of osteogenic sarcoma were examined. The vascularity of the osteoblastic form of these tumors varied with the degree of malignancy. Most, but not all, of the tumors contained very malignant, highly vascular parts which could be demonstrated angiographically, and which provided confirmation of the diagnosis. Different forms of osteogenic sarcoma were found to display microangiographic differences in structure.

ZUSAMMENFASSUNG

Vierundzwanzig Fälle von osteogenem Sarkom wurden untersucht. Die Vaskularisation des osteoblastischen Typus dieser Tumoren variierte mit dem Grad der Malignität. Die meisten Tumore, doch nicht alle, enthielten hochmaligne, stark vaskularisierte Partien, die angiographisch demonstriert werden konnten und die Diagnose bestärkten. Es wurde gefunden, dass verschiedene Formen von osteogenen Sarkomen eine verschiedene mikroangiographische Struktur haben.

RÉSUMÉ

Les auteurs ont examiné vingt-quatre cas de sarcome ostéogénique. Le caractère vasculaire de la forme ostéoblastique de ces tumeurs varie suivant le degré de malignité. La plupart, mais non la totalité, de ces tumeurs comprenait des parties très malignes, hautement vasculaires, qui ont pu être mises en évidence par l'angiographie et qui ont apporté une confirmation au diagnostic. Les auteurs ont constaté que des formes différentes de sarcomes ostéogéniques présentent des différences microangiographiques de structure.

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MEDIONECROTIC DISSECTING ANEURYSM OF THE ABDOMINAL AORTA

Report of a case diagnosed by means of aortography

by

HANS LUDIN, PETER WAIBEL and SIEGFRIED SCHEIDEGGER

Dissection of the walls of the aorta or peripheral arteries may result from arteriosclerosis, medionecrosis, or trauma such as the intramural injection of contrast media in lumbar aortography (WOLFMAN and BOBLITT).

Medionecrosis of the aorta occurs much less often than arteriosclerosis but, when it exists, frequently causes dissection. Its primary site of predilection is the ascending aorta. Dissecting medionecrosis confined to the abdominal aorta and its ramifications is so rare that KNUTSSON (1959) wrote: 'Medionecrosis aortae idiopathica ... does not appear in the abdominal aorta'. We have, however, had the opportunity of establishing the diagnosis of such a condition by aortography prior to operation and ante mortem and feel justified in reporting our case in detail.

A woman, aged 66, with no significant history apart from slight systolic hypertension of 160 mm Hg and some dyspnoea on exertion, suddenly experienced severe lumbar pain, radiating over the abdomen; the symptoms gradually subsided. The next day similar pain, but of increasing severity, caused her to collapse; the pain then gradually subsided. Four days later, following a further attack, she was admitted to hospital. On examination her blood pressure was 55/25; she was somnolent and in poor general condition. There was no abdominal rigidity; the legs were somewhat oedematous but not cyanosed. Following blood transfusion, the blood pressure rose to 165/95 and a strong right-sided paravertebral pulsation

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a b

Fig. 1. Retrograde aortograms in supine and right posterior oblique positions. The dissection channel is projected to the left of the aortic outline.

could be felt on abdominal palpation. Despite numerous transfusions the haematocrit values dropped continuously, yet only a trace of occult blood was present in the faeces. A tentative clinical diagnosis of retroperitoneal haemorrhage from an abdominal aortic aneurysm was made.

On retrograde aortography it proved impossible to pass the catheter up through the upper part of the right or left iliac arteries; 40 ml Urografin 76 % were therefore injected under high pressure and after compressing both femoral arteries with the tip of the catheter placed near the origin of the left hypogastric artery. The patient collapsed repeatedly during this procedure and was treated with further transfusions. Apparently because of the low blood pressure the contrast medium could be forced up the abdominal aorta to the level of the renal arteries. The same quantity of contrast medium was again injected with the patient in the left posterior oblique position.

The examination showed (Fig. 1) marked displacement of a dilated portion of the abdominal aorta to the right, up to the level of L 1—L 2, a few centimeters below the origin

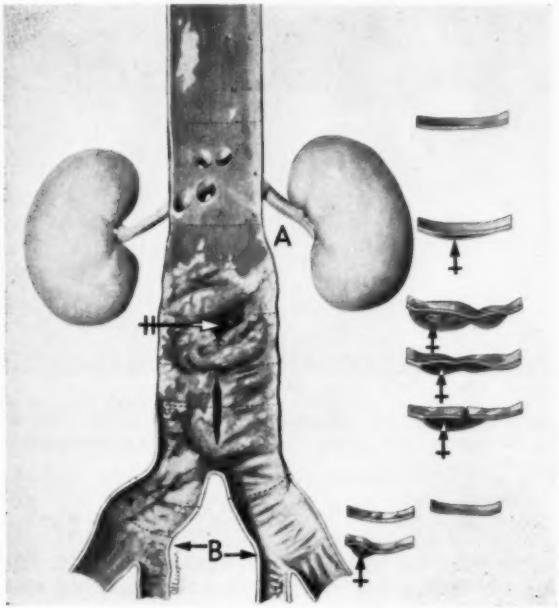

Fig. 2. Autopsy specimen opened from behind. The vessels are widened between (A) and (B). The dissection channel (‡) is indicated at the cross sections seen to the right; these correspond to the various levels of the aortic wall indicated by striped lines. Transverse ruptures of the inner layers of the aortic wall (⇔).

of the renal arteries which were not filled. The aortic segment above the abruptly ending dilatation had, except for its S-shaped connection with the aneurysm, a normal left parasagittal position, a normal calibre and smooth contours. The aortic bifurcation was displaced to the right and posteriorly. Whereas the contours of the dilated portion of the abdominal aorta were quite smooth on the right side, a 7 to 8 cm wide dissection channel was visible in front of and slightly to the left of the aortic lumen, reaching from the vicinity of the upper limit of the dilatation down to the left common iliac artery just proximal to the origin of the left hypogastric artery. The velocity of flow within the dissection channel and the main vessel was about equal. Additional films obtained after a few minutes revealed a left paravertebral retroperitoneal contrast extravasate which displaced the left psoas muscle and ureter laterally. The diagnosis of a dissecting aneurysm of the abdominal aorta with retroperitoneal bleeding was established. Better knowledge of the pathology of dissecting aneurysms would probably have permitted the diagnosis of medionecrosis to have been made from the smoothness of the aneurysmal walls and the normal appearance of the upper portion of the abdominal aorta and the lower iliac and femoral arteries.

A few hours later an anterior external rupture of the aorta was sutured. The patient recovered satisfactorily after the operation and further transfusions. Nine days after operation a slight icteric discoloration and a moderate azotaemia appeared, diminishing, however,

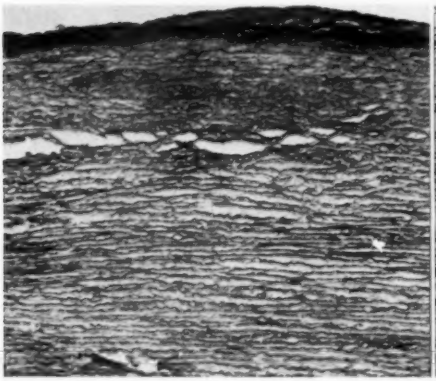

Fig. 3. Idiopathic medionecrosis of the aorta. Numerous cystic spaces of the media with mucoid degeneration.

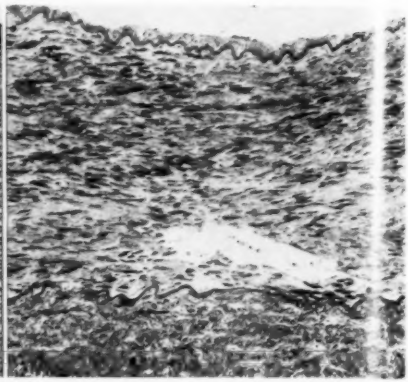

Fig. 4. Large cystic space in the media with mucoid degeneration and diffuse fibrosis in media and adventitia.

during the following days. These signs increased again two weeks after operation, and the urine contained some casts indicating kidney damage. Some days later another attack of shock and collapse occurred, and a haemorrhagic diathesis developed. After an attempt at treatment by dialysis the patient died with pulmonary oedema on the nineteenth day after operation.

Autopsy confirmed the radiologic findings. Fig. 2 is a drawing of the aorta opened from behind and the findings at cross sections through the aorta and iliac arteries at various levels. The sudden increase of the aortic circumference by 2.5 cm about two centimeters below the origin of the renal arteries is clearly demonstrated (arrow A); the widened portion reaches down to the common iliac arteries (arrow B). The dissection channel extends from the upper end of the aneurysm down to the lower extremity of the diseased vascular segment in the left common iliac artery (see cross sections ↔). The involved vessels had lost their normal resilience; some slight and localized atheromatous changes were found within the thoracic aorta. The first transverse rupture of the inner layers of the aortic wall proved histologically to be quite recent and may have occurred when the first symptoms appeared. The complete external rupture of the aortic wall, measuring 3.3 cm in length, was partially closed by sutures. The wall had apparently ruptured beyond the sutured area during the last attack of collapse and shock. Histologic examination suggested the complete rupture to be more recent than the inner rupture.

The retroperitoneal and pelvic connective tissue was infiltrated by relatively fresh blood. Both renal arteries were intact. The small and large bowel showed extensive haemorrhagic infarctions with slight paralytic ileus evidently due to partial occlusion of the mesenteric arteries caused by the aortic dissection.

Histologic examination of the wall of the diseased portion of the abdominal aorta and common iliac arteries revealed the presence of a typical medionecrosis, usually to be found in cases of dissecting aneurysms of the aortic arch. The atheromatous changes and fibrosis of the intimal layer appeared less in degree and extent than usually seen in cases of the same age and sex. A new intima had not yet formed in the dissection channel situated within the necrotic media (Figs 3 and 4). A few vasa vasorum showed a moderate proliferation of the intima and some small round cell infiltration of the adventitia.

Discussion

Dissection of the arterial wall in arteriosclerosis, mostly quite limited in extent, is caused by ulceration of the intima and media. Atherosclerosis with complicating aneurysmal dilatation and dissection is a disease affecting older age groups. Among 15 autopsy cases of dissecting aneurysms reported by LODWICK, 12 were of the 'arteriosclerotic' type, mostly situated in the lower abdominal aorta and most of them asymptomatic. The average age was over 70 years.

Medionecrotic dissecting aneurysms, however, occur far less frequently. According to SAILER they are detected in about 0.25 per cent of autopsy cases, whereas MANIGLIA and GREGORY have found arteriosclerotic aortic aneurysms in one per cent of 1 000 autopsies, 80 per cent of these aneurysms being situated in the abdominal aorta. SHENNAN states that most medionecrotic aneurysms occur between the fourth and the seventh decades. The average age of three cases reported by LODWICK was 33 years. According to SHENNAN medionecrosis occurs not infrequently at younger ages and even in infancy. The primary lesion is situated almost exclusively in the ascending aorta and the aortic arch. The aneurysms often dissect into the brachiocephalic arteries and, in about 40 per cent of cases, down into the lumbar aorta or even into the iliac arteries (MOTE and CARR). In one case described by LODWICK three separate dissected portions, situated in the proximal and distal part of the aortic arch and below the origin of the renal arteries, were found. SHENNAN reported dissections to be multiple in about 10 per cent of cases.

KAUFMANN describes the autopsy findings in a young female with annular dissection of the lumbar aorta extending down to the iliac arteries and fatal haemorrhage due to external rupture. Histologic findings are not reported, but the young age suggests the lesion to have been of the medionecrotic type. One case reported by JOHNS showed much similarity to ours; the internal rupture was situated just below the inferior mesenteric artery, and the external perforation slightly below the inner one. The external perforation was sutured, but the patient died on the eighth postoperative day because of renal failure due to nephrosis. Autopsy revealed the lesion to be of the medionecrotic type; an exact preoperative diagnosis was not established.

It appears that the cases described by DE ANGELIS, STAEMMLER, SZILAGYI and EYLER were of an arteriosclerotic nature. The only case in which a preoperative aortographic diagnosis of the condition has been established seems to be the one reported by EYLER, probably identical with the one discussed by SZILAGYI, in which the aneurysm was resected and successfully replaced by a graft. Further cases have not been found in the literature.

The aetiology and pathogenesis of medionecrotic aneurysms is not well understood for it is not clear whether the rupture of the intima and the adjacent inner two-thirds of the media originates from the lumen or from the layer

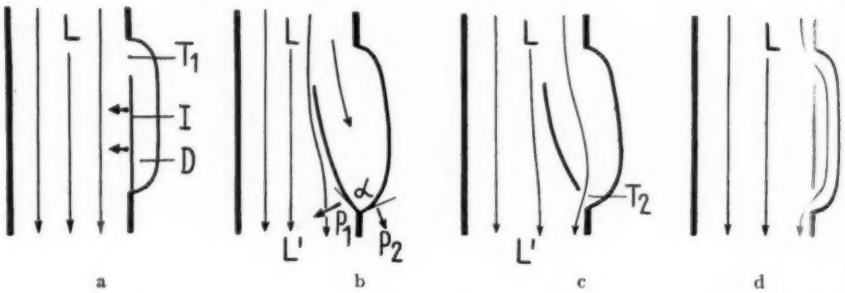

Fig. 5. Pathogenesis of extension of aortic wall dissection. D — Dissection channel, I — Flap, L — Aortic lumen above flap, L — Aortic lumen below flap, P_1 and P_2 — Blood pressure components, T_1 — Primary transverse tear, T_2 — Secondary transverse tear.

in which dissection takes place. According to the latter hypothesis (KRUKENBERG, SAILER, KAUFMANN) a mural haematoma would first develop, probably from some lesion of the vasa vasorum. Repeated paroxysmal extension of the dissection may be explained as follows.

Blood entering the dissection channel D (Fig. 5) from the aortic lumen L through a transverse tear T_1 mobilizes an inner flap I. Blood flowing within L will exert a systolic pull of about 2 to 3 g/cm² and a diastolic pull of about 0.5 g/cm² on the flap, as calculated from Bernoulli's formula, the pull being a quadratic function of blood velocity. It remains speculative whether turbulence will occur, but probably some additional haemodynamic pressure, due to the prominence of the flap into the lumen, will enhance the oscillatory central displacement of the flap, depending on flap mobility, blood flow, and the aortic dimension. It appears questionable whether such forces are sufficient to produce slow extension of the dissection. Increased blood flow, however, due to physical exertion etc, may cause the flap to move towards the opposite aortic wall to such an extent that blood flow along the flap is markedly diminished, and blood pressure below the flap in L′ is lowered. This causes the flap to be pressed towards the opposite wall, blocking the blood flow. The entire aortic blood pressure is exerted on the walls of the dissection channel D, thus also on its distal extremity with components P_1 and P_2 at an angle of $180° - \alpha$, producing a sudden and rapidly progressive distal dissection, mostly followed by re-entry or external rupture according to the local pathology. This explains why intercostal and lumbar arteries are not infrequently torn across. When a re-entry T_2 is established, either spontaneously or by operative fenestration (DE BAKEY), rising blood pressure in L′ causes the flap to move back (Fig. 5 c) and blood will then flow through L and D (Fig. 5 d), the blood pressure being about equal in both lumina.

This hypothesis explains the observation of LODWICK in one of his cases of a medionecrotic type in which cyanosis and ischaemia of the lower part of the body disappeared immediately as soon as re-entry took place. Thus the establishment of a re-entry prevents external perforation and progress of dissection, not by lowering the blood pressure within the dissection channel D, but by stabilizing the status quo. In arteriosclerosis the inner flap will be more rigid and the media more effective at resisting dissection than in medionecrosis, thus explaining the lesser extent of dissection in the former condition.

Symptoms are caused by partial or total obliteration of the lumen of the aorta (see above), of its branches by compressing their origin (spinal cord and cerebral ischaemia, haemorrhagic infarction of the intestines, renal damage), but mainly by acute or subacute massive haemorrhage due to external perforation. According to the location and severity of the haemorrhage the main lesion will be situated in the central nervous system (cerebral ischaemia), the pericardium (cardiac tamponade, coronary insufficiency), or the kidneys (shock kidneys).

Death follows the onset of symptoms at greatly varying intervals, depending on the above factors (JONES and LANGLEY, SHENNAN).

In the surgical treatment the following techniques are employed:

1. Resection and grafting, resulting, when feasible and successful, in permanent cure.
2. Suture of the primary intimal tear in cases without re-entry. The tear is often very difficult to locate even at autopsy (SHENNAN, WARREN, BECKWITH and MULLER).
3. Fenestration (DE BAKEY) in cases without re-entry (see above).
4. Reinforcement of the aortic wall by covering it with orlon tissue (WARREN, BECKWITH and MULLER).
5. Fenestration combined with resection and grafting of the aortic portion distal to the fenestration (WARREN, BECKWITH and MULLER).
6. Suture of the external rupture — merely symptomatic treatment.

Operative results depend on the location and nature of the lesion and the procedure employed: In DE BAKEY's material there were 7 survivors out of 10 patients, whereas WARREN, BECKWITH and MULLER reported only one survivor out of 5 patients. Recurrent bleeding and renal failure seem to be the most important causes of postoperative death. Lesions of the abdominal aorta probably have a better prognosis than those involving the aortic arch; they do not cause cardiac tamponade and are easier to detect clinically and radiologically from unexplained blood loss, lumbar pain, abdominal signs, and lateral displacement of the psoas muscle and ureter. The dilated aortic portion can often be palpated. The abdominal aorta may be clamped off distal to the renal arteries for a period sufficient to permit resection and grafting, which is evidently the ideal procedure.

Diagnostic radiology can contribute important information for the evaluation of the local pathology underlying the clinical picture; it can also afford confirmation of the diagnosis of retroperitoneal haemorrhage and angiographic demonstration of the nature and extent of the lesion. Injection of contrast medium well above the proximal end of the aneurysm will demonstrate or

preclude the presence of an open dissection channel and exclude the possibility of an abdominal extension of a thoracic dissection. Even a closed dissection channel may be detectable if the aorta shows a markedly unequal diameter in lateral or oblique views or if the aortic wall is rather smooth or the upper limit of the narrow portion is abrupt. Irregularity of the outline of adjacent vessels would rather suggest an arteriosclerotic basis. It is evident that high retrograde aortography, when feasible, is the procedure of choice provided the catheterisation is performed very carefully.

Acknowledgement

We wish to thank Prof. H. Heusser, Director of the Second Surgical Department of Basle University Hospital, and his co-workers for referring the patient and for the clinical records.

SUMMARY

A case of medionecrotic dissecting aneurysm of the abdominal aorta has been diagnosed by means of retrograde percutaneous aortography. The possibilities of angiographic diagnosis, the differences between arteriosclerotic and medionecrotic dissecting aneurysms, the symptomatology and therapeutic procedures, together with an hypothesis on the mechanics of the extension of dissections, are discussed.

ZUSAMMENFASSUNG

Ein Fall von medionekrotischem dissezierendem Aneurysma der Abdominalaorta wurde mittels retrograder, perkutaner Aortographie diagnostiziert. Die Möglichkeiten der angiographischen Diagnosestellung, die Unterschiede zwischen arteriosklerotischen und medionekrotischen dissezierenden Aneurysmen, die Symptomatologie und Therapie sowie eine Hypothese über die Mechanik der Ausbreitung der dissezierenden Veränderungen werden besprochen.

RÉSUMÉ

Un cas d'anévrisme disséquant par nécrose de la media de l'aorte abdominale a été diagnostiqué par aortographie rétrograde percutanée. Les auteurs étudient les possibilités du diagnostic angiographique, les différences entre les anévrismes disséquants artérioscléreux et médionécrotique, la symptomatologie et les traitements, ainsi qu'une hypothèse sur le mécanisme de l'extension du processus disséquant.

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PANTOPAQUE MYELOGRAPHY IN AVULSION OF THE BRACHIAL PLEXUS

by

JACK LESTER

A tearing away of the nerve roots from the medulla spinalis in the brachial plexus produces a clinical picture which, although characteristic, cannot definitely be distinguished without the aid of myelography from more peripheral lesions of the corresponding nerve roots. Typical myelographic appearances in these lesions were first described in 1947 by MURPHY, HARTUNG, and KIRKLIN who, by 1949, had encountered 7 cases. In 1954, WHITELEATHER of the same clinic reported 8 cases, 4 of which had, however, already been mentioned in 1949. Similar cases have been published by JAEGER and WHITELEY (1953), TARLOV and DAY (1954), WHITE and HANELIN (1954), RAYLE et coll. (1955), WIEDENMANN and DECKER (1956), BORELLI and MAGLIONE (1956), SASSAROLI and SPACCARELLI (1957), PASSARINI (1959), and RASMUSSEN (1959). In all, 33 cases of brachial plexus avulsion diagnosed by pantopaque myelography have been reported; 12 of these have been confirmed at operation.

These avulsions arise as a result of severe injury to the shoulder region, usually after a motor accident or a fall from a height. Multiple fractures and sometimes cranial trauma with loss of consciousness are often present as well. In lesion of the uppermost roots (C5, to C7) Erb-Duchenne paralysis, affecting mainly the shoulder movements and elbow flexion, may occur. Damage of the lower roots (C8 and Th1) produces Klumpke's paralysis, which chiefly involves the muscles of the hand. If Th1 has been torn away Horner's syndrome is also evident, owing to injury of the sympathetic fibres to the stellate ganglion.

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accid paralysis with corresponding impairment of the sensory function usually sets in at once; as the sensory nerve roots are thicker and hence have greater powers of resistance than the motor roots (TARLOV) the sensory impairment may, however, be less extensive. Burning or darting pain along the affected nerve pathways is often reported.

A tentative diagnosis may be made by spinal puncture, the cerebrospinal fluid usually being blood-streaked during the first 5 to 6 weeks. Electromyography is another important aid in indicating the extent of the damage.

Exact clinical localization of the nerve damage is often difficult owing to masking by injuries of the soft tissue and bone. However, a lesion of the dorsal scapular and long thoracic nerves (paresis of the rhomboideus, levator scapulae, and serratus anterior muscles) and a lesion of the sympathetic fibres in Th1 (Horner's syndrome) indicate that the nerves have been damaged either before or directly after their emergence from the intervertebral foramina, and hence suggest that avulsion has occurred. Signs of injury to the medulla spinalis also point to the same possibility (JAEGER and WHITELEY).

As a result of violent trauma the dura mater and the arachnoidea which encircle the roots as far as the foramina may become torn. The dura mater is the stronger of the two membranes, while the arachnoid membrane and the slender radicular fibres are easily damaged following damage to the former; the cerebrospinal fluid then leaks out into the space formed when the peripheral nerve stump retracts. The dura and arachnoidea gradually proliferate and close this space to produce a kind of traumatic meningocele (WHITE and HANELIN), a feature underlying the characteristic myelographic appearances.

Myelography with iodized oil (Pantopaque) reveals in typical cases that the contrast medium runs outside the vertebral canal towards the intervertebral foramina and collects in diverticulum-like pockets corresponding to the avulsed roots. The myelographic examination should be carried out with the patient in the prone position, when the opening of the pockets lies at the lowest level. At least 6, preferably 9 ml, of contrast medium should be used, as filling of the pockets takes some time and may fail to occur if too small an amount of medium passes too rapidly. WHITE and HANELIN, for instance, observed defective filling of a pocket in one of their cases, and found obstructing adhesions at subsequent exploration.

The avulsions may have two different myelographic appearances, but both are often seen in one and the same case: (1) contrast-filled extradural pockets ranging in size from a pea to a walnut and extending obliquely and caudally from the vertebral canal to the foramina (Figs 1, 2, and 3); (2) less marked changes in the lateral outline of the contrast medium at the site of the affected spinal nerves. The outline may either be convex medially (TARLOV), irregular and wavy (BORELLI), or, as we have observed in several cases, show a slight bulge with the convexity laterally. In all cases absence of the usual rounded defect in the contrast filling at the exit site of the spinal nerves is a charac-

Fig. 1. Case 1. Typical large Pantopaque-filled avulsion pocket at the level of the left C 8 and a slightly smaller pocket at Th 1.

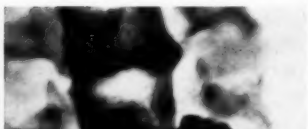

Fig. 2. Case 2. Typical avulsion pocket at left C 7. Small bulge and laterally directed convexity at level of C 6. Normal root outlines absent; asymmetry.

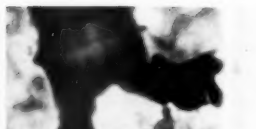

Fig. 3. Case 3. Large avulsion pocket at the site of left C 6. Irregular defect in contrast outline at C 7, probably indicating lesion of the medulla spinalis, and absence of the normal defect corresponding to the nerve root.

teristic feature. A comparison with the sound side shows the condition to be asymmetric (Figs 4 and 5).

Roentgen appearances resembling an avulsion are seen only in the rare extradural arachnoid cysts. These are, however, most commonly found in the thoracic region in association with Scheuermann's disease (Good et coll. 1944); they have been observed in the cervical region in only 3 cases (Meredith 1940, Pendergrass et coll. 1956, and Rayle et coll.). Differential diagnostic problems can hardly arise, however, because the anamnesis and the absence of relevant neurologic signs and symptoms exclude an avulsion.

The prognosis is obviously poor in these avulsions. No appreciable regression, either spontaneous or following a neurosurgical operation, can be expected. The most suitable form of treatment is physiotherapy; movement therapy to keep the joints mobile and myotensor treatment to maintain muscle function. Orthopaedic intervention, or amputation, may have to be undertaken at a later stage.

Material

We have established with the aid of pantopaque myelography the presence of one or more avulsions in 6 patients within the course of six months. These consisted of 5 men and one woman ranging in age from 19 to 39 years. In 5 of the cases the condition was the result of a motor accident while the sixth patient with a fractured scapula had caught his arm in a winch on a fishing boat. Five of the 6 patients had various other fractures and 3 had had a head injury with loss of consciousness. Pantopaque myelography was undertaken

in these 6 cases 14 days to 5 months after the accident. In 3 patients who were examined two, two and a half, and six weeks respectively after the injury slightly blood-streaked cerebrospinal fluid was present; in the others, including one patient examined 18 days after the accident, the fluid was clear.

Clinically, 2 patients had Erb-Duchenne paralysis; avulsion of C 5 and C 6 was demonstrated in one of them and of C 6 and C 7 in the other. The former had paresis of the diaphragm as well; this was not observed in any of the other patients. In 3 patients with Klumpke's paralysis and Horner's syndrome, avulsion of C 8 and Th 1 was noted. The sixth patient had completely flaccid paralysis of one arm and Horner's syndrome; myelography revealed avulsion of C 7 and C 8, and Th 1.

After the diagnosis had been established by myelography none of the patients were submitted to operation but were advised to undergo physiotherapy or orthopaedic treatment.

Case reports

Case 1. Male, aged 24, with paresis of both arms and the left leg immediately after a motor accident. Contusion of the medulla spinalis and fracture of the third thoracic vertebra were diagnosed at another hospital. Regression of the paresis of the right arm and left leg soon occurred but the left arm did not improve. Six months after the trauma he had left-sided miosis, slight diffuse weakness of the right arm and left leg, and severe paresis of the left arm with dystrophy of the hand. Pantopaque myelography (clear spinal fluid) revealed a large typical avulsion pocket filled with contrast medium at the left C 8 and a smaller pocket at the level of Th 1 (Fig. 1). Roentgen diagnosis: Avulsion of C 8 and Th 1 on the left side.

Case 2. Male, aged 19, with paresis of the shoulder and elbow immediately after a motorcycle accident involving trauma to the left shoulder. One month later, he had total paralysis of the deltoid and pectoralis muscles with anaesthesia and analgesia of the corresponding areas; atrophy of the left deltoid and infraspinatus muscles as well as of the muscles of the upper arm was also noted. As suboccipital air myelography (slightly blood-streaked spinal fluid) suggested avulsion of the left C 7, pantopaque myelography was performed and revealed a typical avulsion cyst at this site. A small bulge in the contrast outline, with the convexity laterally, was visible at the level of C 6, and at the same time the normal outline of the root was absent; in other words, there was definite asymmetry as compared with the right side (Fig. 2). Roentgen diagnosis: Avulsion of the left C 6 and C 7.

Case 3. Female, aged 39, had been admitted to another hospital after a motorcycle accident involving loss of consciousness for a few minutes followed by severe paresis of the left arm. She also had fractures of the left transverse processes of the seventh cervical and first thoracic vertebrae. A fortnight later, paralysis of the shoulder movements, severe paresis of the extension and flexion movements of the elbow, and partial paresis of the hand muscles persisted on the left side, as well as analgesia on the dorsal aspect of the left shoulder. Roentgen examination of the thorax revealed that the left side of the diaphragm was raised. Myelography (clear spinal fluid) showed a large avulsion pocket at the site of the left C 6; a large irregular defect in the contrast filling was seen at the level of C 7, and the normal contrast defect corresponding to the nerve root was not visible (Fig. 3). Roentgen diagnosis: Avulsion of the roots of C 6 and C 7 on the left side with probable lesion of the medulla spinalis at C 7.

Fig. 4. Case 5. Small bulges in the contrast outline and no signs of root contours at the right C 8 and Th 1. (Cf. normal root contours on left side.)

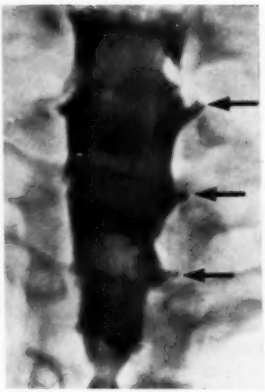

Fig. 5. Case 6. Small lateral projections from the contrast outline, and no signs of root contours at level of the left C 7, C 8 and Th 1.

Fig. 6. Extradural arachnoidal cyst in a man, aged 53, without clinical signs of avulsion. Small contrast-filled pocket at the right C 8 but directly above it the defect corresponding to the nerve root is intact.

Case 4. Male, aged 28, had caught his right arm in the winch of a fishing boat resulting in fracture of the neck of the scapula. When admitted 8 days later, flaccid paralysis of the right upper arm and Horner's syndrome were noted. Suboccipital air myelography (slightly blood-streaked spinal fluid) suggested an air-filled avulsion cyst at the site of the right C 8, and pantopaque myelography revealed a large typical avulsion pocket at the right C 8 and absence of a contrast defect at the root of Th 1. Roentgen diagnosis: Avulsion of the right C 8 and Th 1.

Case 5. Male, aged 24, who was admitted with a fracture of the lower jaw and paralysis of the right arm following a motorcycle accident. One month later, he had paralysis of the right lower arm and hand with areflexia, anaesthesia of the lower part of the arm, and Horner's syndrome. Myelography (clear spinal fluid) revealed small bulges in the contrast outline and absence of the root contours at the level of the right C 8 and Th 1 (Fig. 4). Roentgen diagnosis: Avulsion of the right C 8 and Th 1.

Case 6. Male, aged 22, admitted after a motorcycle accident involving loss of consciousness and fracture of the left lower arm with diffuse paresis and corresponding anaesthesia and analgesia of the entire left arm and Horner's syndrome. Pantopaque myelography performed 18 days later yielded clear spinal fluid; small bulges in the contrast outline, which was convex laterally, were present, and there was no evidence of the root outline at the level of C 7, C 8 and Th 1 (Fig. 5). Roentgen diagnosis: Avulsion of the left C 7, C 8 and Th 1.

We have also recently demonstrated by pantopaque myelography an extradural arachnoidal cyst which, at first glance, resembled an avulsion-pocket. The patient was a 53-year-old man with myelopathy of unknown origin. He had no signs or symptoms referable to the upper extremities and had never

ha any injury to the right shoulder. A pocket was visible at the level of the rig C 8, but directly cranial to it the defect in the contrast outline at the site of the nerve root was intact (Fig. 6).

Discussion and Conclusions

I most of the earlier publications main emphasis has been laid on large con ast-filled extradural pockets. A few remaining nerve fibres are sometimes fou d in these at operation and hence there is a possibility, although as a rul very slight, that regression of the paralysis may take place.

Most patients, however, have in addition, or even exclusively as in our Cas s 5 and 6, less marked but equally pathognomonic changes in the root pockets; the characteristic feature consists in the absence of the central defect in the contrast medium corresponding to the position of origin of the nerve root. That an avulsion is also present in these cases may be regarded as certain, seeing that 7 out of the 12 cases proved by operation (TARLOV's cases 1 and 2, WHITELEATHER's cases 1 and 2, and BORELLI's three cases) displayed this myelographic appearance. TARLOV and WHITELEATHER considered that absence of contrast-filling in a large pocket is due to the lesion of the dura mater having healed, since they had noted after laminectomy, in addition to a complete avulsion in which not a single nerve fibre remained, that the dura lesion was replaced by dense scar tissue. Other investigators, such as RAYLE, held that the atypical myelographic appearances may be explained as avulsion without a dura lesion, but no operative confirmation of this seems to have been reported.

It is thus obvious that a diagnosis of avulsion can only be made by careful pantopaque myelography with clear demonstration of all the root pockets belonging to the brachial plexus. Air myelography is capable of demonstrating large contrast-filled pockets, but less noticeable changes in the root pockets cannot be revealed with this technique.

Roentgenographic demonstration of avulsions of the brachial plexus is of great practical value as it allows an immediate assessment of the prognosis and serves as guidance for correct treatment. If the lesions can be established, the patient will be spared useless surgical intervention, and no particular spontaneous regeneration can be expected. Pantopaque myelography is therefore indicated in all cases in which the condition is suggested.

SUMMARY

The myelographic appearances in 6 cases of avulsion of the brachial plexus are described. In addition to the typical extradural contrast-filled pockets, less marked changes around the nerve roots are often discernible, the characteristic feature being absence of the normal defect in the contrast medium at the point of exit of the nerve root. These changes are equally pathognomonic as the former signs and can only be demonstrated by pantopaque myelography.

ZUSAMMENFASSUNG

Das myelographische Bild von 6 Fällen von Abrissen des Plexus brachialis wird beschrieben. Ausser den typischen kontrastgefüllten Extraduraltaschen sieht man oft kleinere Veränderungen in der Umgebung der Nervenwurzeln, wobei das charakteristische Bild der normalen Kontrastmittelaussparung an der Austrittsstelle der Nervenwurzeln fehlt. Diese Veränderungen sind ebenso als pathognomonisch anzusehen wie die erstgenannten Symptome und können nur mittels Pantopaque-Myelographie nachgewiesen werden.

RÉSUMÉ

L'auteur décrit les signes myélographiques dans 6 cas d'arrachement du plexus brachial. Outre les poches extra-durales typiques remplies par le contraste, on observe souvent des modifications moins importantes autour des racines nerveuses, caractérisées par l'absence de la lacune normale dans le moyen de contraste au point de sortie de la racine. Ce dernier signe est aussi pathognomonique que le premier, et ne peut être mis en évidence que par myélographie au pantopaque.

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EXPERIMENTS WITH CHOLECYSTOKININ

by

ALDO TORSOLI, MARIA LETIZIA RAMORINO, CORRADO COLAGRANDE and GIAMPIERO DEMAIO

Cholecystokinin is a hormone that promotes gallbladder contraction and belongs to the group of 'silent hormones' (so called by JORPES) that control digestion in the upper part of the gastro-intestinal apparatus.

IVY and OLDBERG first provided evidence that the cholecystokinetic effect of duodenal mucosa extracts is due to a specific hormone, of which they obtained the first samples. But IVY and OLDBERG's cholecystokinin, as well as pancreozymin found many years later by some English workers (DUNCAN et coll.), turned out to be of little practical value; the preparations moreover caused undesirable side effects. In 1954, JORPES and MUTT finally elaborated a method of obtaining highly purified and active preparations, suitable for clinical purposes. The biologic assay of the hormone is made according to IVY and OLDBERG or by closely related methods. The IVY dog-unit is defined as the amount of dried material, free of vasodilatatory effects, which, when dissolved in normal saline solution and injected intravenously within 10 or 15 sec, causes within the first three minutes an intra-gallbladder pressure rise of 1 cm. The cholecystokinetic effect of cholecystokinin is demonstrated in vivo as well as in vitro.

The release of the hormone from the duodenal mucosa seems to be connected with the stimulation of a procainsensitive receptor which is connected to the cells producing cholecystokinin by a pathway that can be blocked by hexamethonium (HONG, MAGEE and CREWDSON 1956).

Cholecystokinin also causes the relaxation of the sphincter of Oddi; an effect

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of contraction on this structure, reported by some authors to occur with the crude preparations, is probably due to the presence of a factor different from the hormone which has been proved to produce intestinal contraction (P substance). It was as a matter of fact shown that in vitro purified cholecystokinin preparations have an effect on the gallbladder but not on the intestine of the guinea pig, while the P substance is active on the intestine but not on the gallbladder (HULTMAN 1955).

The cholecystokinin preparation of JORPES and MUTT was assayed by Ivy and JANECECK (1959) and found to be the most potent preparation ever tested in their laboratory. It contained a small amount of secretin, i. e. from 0.5 to 1 unit of secretin for each unit of cholecystokinin.

Clinical trials with cholecystokinin were first carried out in the Spring of 1955 at Södersjukhuset in Stockholm and a short report on the first results was given by JÖNSSON in August of the same year. Since then many reports have been published on the subject and the reader is referred to the bibliography accompanying this paper.

Materials and Methods

Radiologic examinations after cholecystokin (Cecekin Vitrum) were carried out in 175 cases.

The total human material consisted of 154 subjects (57 men, 97 women) aged from 12 to 76 years; 44 were normal and 110 presented symptoms referable to the biliary tract. The roentgenologic demonstration of the biliary system was obtained by oral and intravenous cholecystography (121 cases), by barium gastro-intestinal examinations (8 subjects with operative choledochoduodenostomy) and by fistulography (25 postoperative examinations).

Routine examinations with serial exposures were used in 56 cases, cinefluorographic methods in 83 cases and cinefluorography combined with simultaneous electromanometric recording of biliary and duodenal intraluminal pressures in 36 cases.

The material was divided into the following groups:

A. One cholecystokinin test (107 cases)
B. Various doses and methods of administration of the hormone (5 cases)
C. Egg-yolk meal and cholecystokinin (20 cases)
D. Egg-yolk meal and various doses and methods of administration of cholecystokinin (22 cases)

The animal material consisted of 21 dogs. The biliary apparatus was filled by intravenous cholecystography in 9 animals and by operative or postoperative cholangiography in 12 animals.

The examinations were carried out by cinefluorography in 9 dogs and by

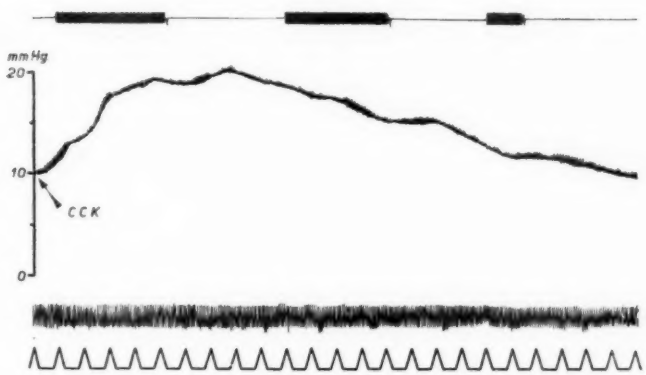


Fig. 1. Intra-gallbladder electromanometric pressure curve after cholecystokinin (75 Ivy units/3 min) following cholecystectomy in man. From top to bottom: synchronization of cinefluorographic exposures, intragallbladder pressure curve, respiration diagram, and time curve (in minutes).

cinefluorography combined with simultaneous electromanometric recording of intraluminal biliary and duodenal pressure in 12 animals.

Cholecystokinin was administered in doses of 1 Ivy unit per kg bodyweight by intravenous injection during 3 min (8 animals), by intravenous perfusion during 30 min (7 animals), and by intravenous injection during 30 sec (6 animals).

The cinefluorographic examinations were carried out with two image amplifiers (Westinghouse and Siemens 5″), equipped respectively with a 16 mm Paillard and a 35 mm Arriflex camera and using Kodak Tri-X negative film. Frames per second: 12, 16 and 24. Electromanometric recordings were made by two Elema strain-gauge manometers connected with a 6-channel recording unit. Radiographic serial exposures were obtained at 1, 2, 4, 8, 12, 18, 24, 30, 40, 60, and 90 min after the beginning of the injection. The largest transverse diameter of the gallbladder was measured for the quantitative evaluation of gallbladder emptying (Brodén). The use of such a comparatively rough method seemed suitable for our purpose in consideration of the large material, which was statistically examined.

Action of cholecystokinin on the gallbladder

The findings show that in human subjects and in the dog cholecystokinin promotes the contraction of the gallbladder and favours its emptying. The effect on the contraction must be distinguished from the degree of emptying; the latter, though a consequence of the former, is not proportional to it but

Fig. 2

Fig. 3 a

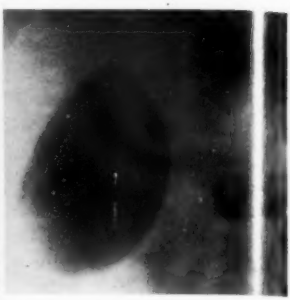


Fig. 3 b

Fig. 2. Cholecystography in a normal case, 4 min after the injection of cholecystokinin (75 Ivy units/3 min). The cystic and common bile ducts, and the choledochoduodenal junction are well shown. The appearance of duodenum is normal.

Fig. 3. Cholecystography in a case with biliary symptoms. a) Before and b) 1 min after beginning of cholecystokinin injection (75 Ivy units/3 min) which caused rapid contraction of the infundibulum; gallbladder became ball-shaped.

presents a large variability depending upon the dose and the method of administration of the hormone and upon the individual reactivity of the infundibulum and cystic duct. The effect upon the contraction, demonstrated by a rising electromanometric curve of intracholecystic pressure, tends to be constant. Cholecystokinin produces an active cholecystic contraction in which, after a short period of latency (10 to 30 sec), two successive periods can be distinguished: a period of tension in which the junction of the gallbladder and cystic dust is closed (15 to 60 sec) and a period of expulsion in which the junction of the gallbladder and cystic duct is open (5 to 30 min). This contraction is of the tonic type; no phasic motor events are recorded (Fig. 1).

The effect on the evacuation is marked by a reduction in the gallbladder size and by filling of the biliary ducts. The emptying is usually accompanied by an early reduction in the transverse diameter of the infundibulum which in most cases becomes clearly outlined. Throughout the first period (10 min) the reduction in the size of the infundibulum tends to prevail over those of the body and fundus of the gallbladder. Alternative contractions and relaxations are evident and have no counterpart in other tracts. The early and conspicuous filling of the cystic duct and, frequently, of the hepatic ducts is due to this dynamic activity of the infundibulum; even the choledochoduodenal junction is clearly visible (Fig. 2). In some cases, however, the dynamic response may be excessive, in which cases the infundibular cavity becomes smaller and eventually disappears; sometimes, after the normal period of tension, the function

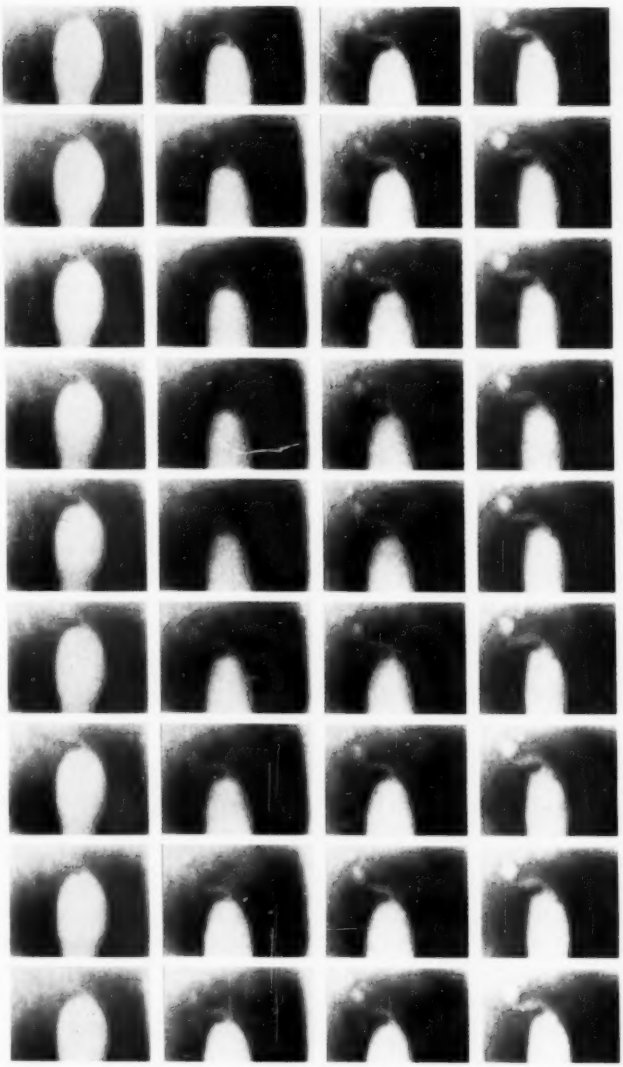


Fig. 4. Cholecystography in a case with biliary symptoms. Effect of amyl-nitrite upon hypertonicity of the infundibulum-cystic duct junction induced by overdosage of cholecystokinin (75 Ivy units/30 sec). (Cineroentgenography.)

a b c

Fig. 5. Cholecystography in a normal case: a) before fatty meal, b) 30 min after fatty meal, and c) 12 min from the beginning of the cholecystokinin injection (75 Ivy units/3 min). Marked contraction of infundibulum.

of the gallbladder and cystic duct remains closed or recloses after a short period of relaxation; in both cases the gallbladder emptying may be partially or completely blocked (Figs 3 and 4).

These events deserve to be compared with those which follow the administration of the usual fatty meal (Fig. 5). Both the period of latency and the periods of tension and expulsion also occur after the egg-yolk meal. The various dynamic events, however, progress very slowly and the maximal evacuation is generally reached after at least one hour; these events are intercoordinated, i. e. the contraction is the result of the balanced participation of the different sectors of the gallbladder (fundus, body and infundibulum). As a rule, the gallbladder is reduced concentrically; the contraction of the infundibulum is evident only at the end of the emptying; the visibility of the ducts is mostly limited to the cystic duct and to part of the common bile duct. A fatty meal therefore seems to be a more physiologic means of emptying the gallbladder than cholecystokinin; it makes it possible in fact gradually to produce almost complete evacuation. Cholecystokinin, on the contrary, is a prompt and efficient stimulant of gallbladder contraction, especially of the infundibulum (Figs 6 and 7).

It is possible to demonstrate a direct relationship between the dynamic response of the infundibulum and of the junction of the gallbladder and cystic duct and the dose (amount and period of administration) of cholecystokinin.

The effects of different doses and different methods of administration were compared in a group of 12 normal subjects and 15 subjects with biliary tract disorders. Our findings may be summarized as follows:

1. In normal subjects and sometimes also in subjects with biliary disease, cholecystokinin can produce obvious contraction even in doses of a half or two-thirds of the standard dose (1 Ivy unit per kg bodyweight injected intra-

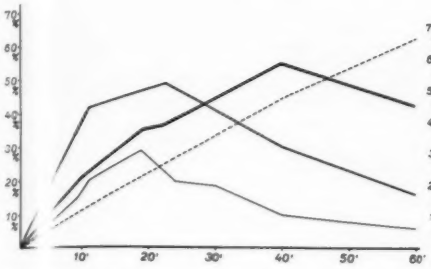


Fig. 6. Gallbladder emptying rate in 42 subjects, some healthy and some with biliary disorders, after fatty meal (interrupted line), and after cholecystokinin injection, 1 Ivy unit/kg bodyweight, in 30 sec (thinnest line), 3 min (medium line), and 30 min (thickest line). After the fatty meal the emptying is more gradual and more complete. When the hormone is administered by perfusion (30 min), the emptying rate is similar to that induced by the fatty meal. The standard cholecystokinin test produces an earlier and shorter gallbladder contraction.

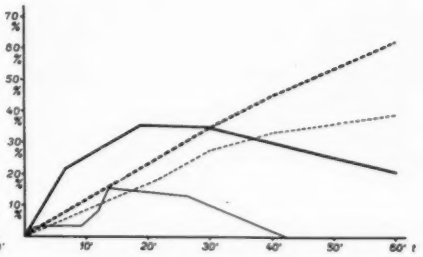


Fig. 7. Mean emptying values obtained in 42 subjects, 16 healthy and 25 with biliary disorders, after fatty meal and injection of cholecystokinin. The heavy interrupted line indicates fatty meal test in normal subjects, and the thinner interrupted line same test in subjects with functional biliary disorders. The heavy continuous line indicates cholecystokinin injection in normal subjects and the thinner continuous line in subjects with functional biliary disorders.

venously in 3 min) or in slowly injected standard doses (0.8 to 2.5 Ivy units per min). In the normal subject a dose of 25 Ivy units injected within 3 min causes an average 40 % emptying of the gallbladder.

2. The dose that assures in a normal subject the highest degree of gradual evacuation is 1 Ivy-unit per kg (dissolved in about 350 ml of physiologic solution) and administered by intravenous perfusion within a period of 30 min. This dose (at the end of the perfusion) causes an evacuation of 50 to 70 %; this form of the cholecystokinin test is the one most similar to the egg-yolk test, from which it, however, differs by a clearer outlining of the infundibulum.

3. The standard dose administered within the standard period may be used as a test for the functional exploration of the infundibulum. In normal subjects this dose produces a definite, but not complete contraction of the infundibulum, allowing the further partial evacuation of the bile from the fundus and from the body of the gallbladder. The gallbladder evacuation, on the whole, reaches an average of about 40 % within 20 min.

4. Greater doses injected within the standard or shorter periods of time induce abnormal hypertonic and spastic reactions of the infundibulum in many normal subjects, thereby arresting the evacuation. The hypertonic and spastic condition may be prevented or at least partially reduced by the administration of amyl nitrite.

Action of cholecystokinin in the common bile duct and the apparatus of Oddi

Both in human subjects and dogs cholecystokinin seems to cause variations in the tone of the wall of the common bile duct and clearly modifies the normal basal dynamic activity of contraction and relaxation of the lower part of the duct. The action of the hormone has also been demonstrated following cholecystectomy; the contraction was sometimes more marked in these cases than in normal cases, a fact which suggests a direct action upon the muscular structure of the duct.

The variations in the shape and pressure of the duct (registered by cinefluorography and by electromanometry respectively, and apparently independent of the bile flow through the duct), have been interpreted as variations in tone of the common duct walls. The main effect of cholecystokinin on the apparatus of Oddi is relaxation (Fig. 8). According to BOYDEN, the apparatus comprises the common channel (extra- and intramural), the pancreatic duct and the common channel musculature; in some cases a common tract of the bile and pancreatic ducts exists. Even when the intramural part is organically obstructed, the relaxation induced by cholecystokinin is still evident in the extra-mural part (Fig. 9). Relaxation sometimes involves opening movements of Oddi's muscles; these movements become more and more frequent and start 30 sec to 1 min after the beginning of the in-

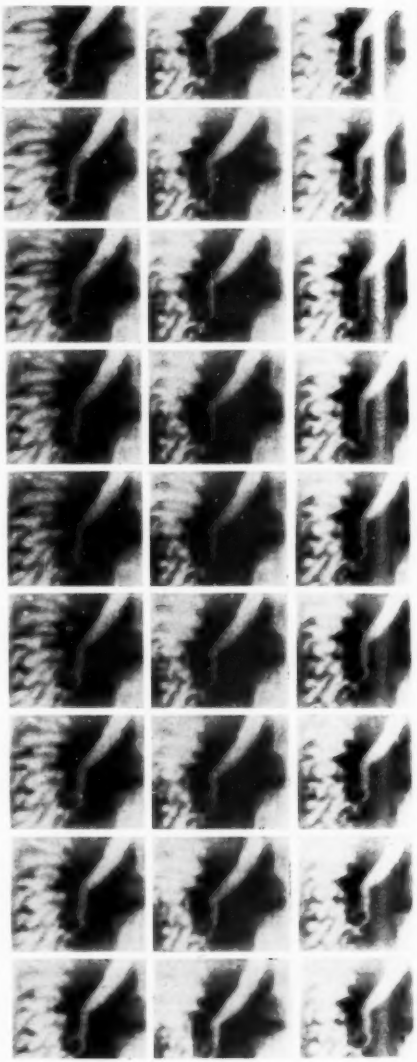
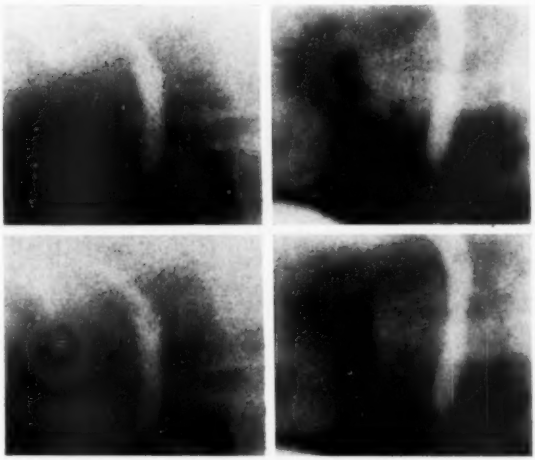
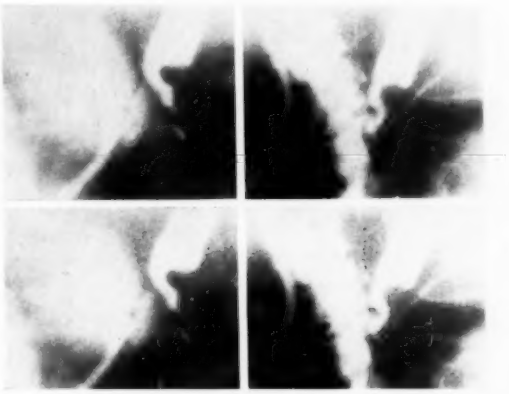


Fig. 8. Choledochoduodenostomy following cholecystectomy. Gastro-intestinal cinefluorographic examination showing effect of relaxation induced by cholecystokinin (75 Ivy units/3 min) on the sphincter of Oddi.



a b

Fig. 9. Choledochoduodenostomy following cholecystectomy. Sclerosing odditis. Gastro-intestinal cinefluorographic examination: a) before and b) 3 min after the beginning of injection of cholecystokinin (75 Ivy units/3 min). Relaxation of upper part of Oddi's apparatus.



a b

Fig. 10. Hypertonicity of the sphincter of Oddi following cholecystectomy. Intracholedochal probe in situ: a) during artificially induced hyperpressure (30 cm H_2O) and b) during normal perfusion pressure (14 cm H_2O) after cholecystokinin (75 Ivy units/3 min). The cinefluorographic frames, corresponding to examinations performed on different days, suggest that cholecystokinin produces bile reflux into the pancreatic duct.

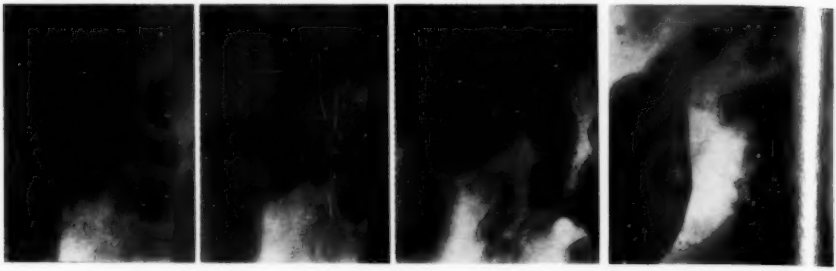


Fig. 11 a Fig. 11 b Fig. 11 c Fig. 12

Fig. 11. Cholegraphy in a case with biliary symptoms. Hypertonic odditis. a) Before, b) 2 min and c) 4 min after the beginning of injection of cholecystokinin (75 Ivy units/3 min). Relaxation of terminal part of common bile duct produced by the hormone.

Fig. 12. Cholecystography in a case with biliary symptoms. The exposure was made 5 min after the injection of cholecystokinin (75 Ivy units/3 min). The contrast medium tends to collect in the superior duodenal loop.

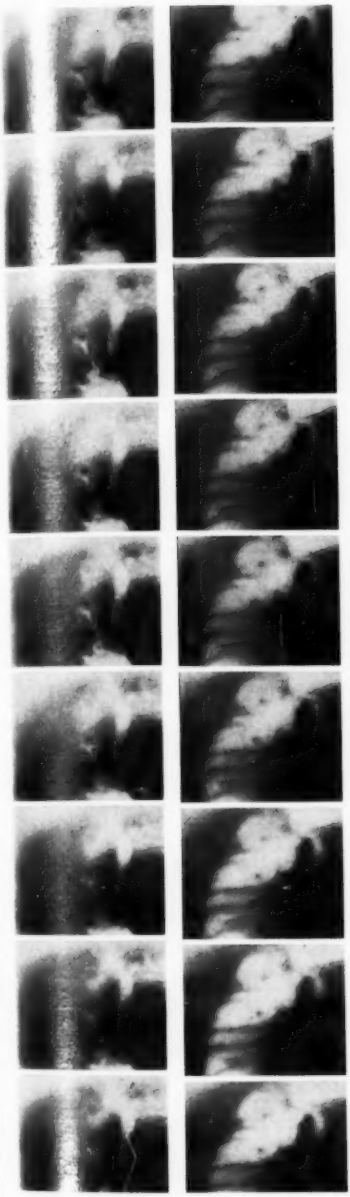
jection. Relaxation is occasionally followed by a period (lasting some minutes) in which the common duct is completely patent.

The fact that in operative cholangiography, with the perfusion pressure constant, cholecystokinin usually produces a pancreatic reflux suggests a direct effect upon the pancreatic duct (Fig. 10). Cholecystokinin may at least temporarily reduce pure Oddi hypertonicity or the functional component of hypertonic odditis (Fig. 11). As demonstrated by JACOBSSON and co-workers, even spasm of the sphincter induced by morphine is relieved by cholecystokinin, which must therefore be considered as the most powerful relaxing agent of the sphincter of Oddi we have to-day.

The reasons why before relaxation the terminal part of the common duct remains closed for about 30 to 60 seconds are not yet understood. There is some evidence that this is only a period of latency of the hormone action, while other experiments indicate that it is a period of active contraction of the sphincter. Present results suggest that an active closure movement may exist when there is a hypertonic condition of the sphincter or when greater or more concentrated amounts of cholecystokinin are used. This subject deserves more adequate studies. It is connected also with the presence in cholecystokinin preparations of slight traces of substance 'P' and of other agents likely to give rise to the motor tonic activity of the duodenum and sphincter of Oddi.

Action of cholecystokinin on the duodenum

Cholecystokinin induces a tonic relaxation of the second part of the duodenum. After the injection of the hormone the duodenal loop assumes unusual radiologic appearances characterized by distension, marked prominence of



the transverse mucosal folds and only occasional peristaltic waves, antiperistaltic waves and pendular movements. The duodenal content tends to collect in the superior duodenal loop and the superior and inferior functional sphincters of the duodenum sometimes have a hypertonic appearance (Figs 12 and 13).

These morphologic and dynamical aspects are quite different from what is generally seen in gastro-intestinal examinations when the duodenum is filled by barium emulsion through the stomach. As shown by the present investigations, these aspects are connected not only with the specific hormonal stimulation but also with the mechanical stimulation of the second part of the duodenum, produced by its filling through the common bile duct. The tonic relaxation comes after a period of latency which is sometimes associated with a transient activation of motility; the same considerations previously made for the activity of the common bile duct hold true for the interpretation of this finding.

Experiments carried out in our laboratory (RAMORINO et coll. unpublished data) suggest that cholecystokinin also stimulates biligenetic activity. The administration of the hormone induces in fact an increase of the bile flow out of a probe inserted in the common duct of cholecystectomized dogs. The increase of flow starts 3 minutes after the beginning of the hormone injection and lasts about 20 minutes. The pH and biliary pigments content of the bile increase after cholecystokinin administration.

Fig. 13. Choledochoduodenostomy following cholecystectomy. Gastro-intestinal cinefluorographic examination: a) before and b) 5 min after beginning of the injection of cholecystokinin (75 Ivy units/3 min). Characteristic appearance of duodenum.

Conclusions

It has been stated by numerous authors that cholecystokinin reduces the time necessary for cholecystographic examinations, and produces good filling of the excretory ducts and, at least in part, facilitates a subsequent roentgenologic examination of the stomach. Our experience, however, suggests that cholecystokinin cannot always take the place of the usual fatty meal. It would appear that the indications for the cholecystokinin hormone and the egg-yolk meal are different and that in many cases the two tests may be integrated.

The administration of cholecystokinin (1 Ivy-unit per kg bodyweight intravenously) is indicated in patients unable to take fats or with obstruction symptoms in the esophagus, stomach, or duodenum or in the study of the functional behaviour of the sphincter of Oddi. A choledochokinetic test to show whether the choledochoduodenal junction is normal, and to show the functional component of a possible narrowing of the sphincter of Oddi, may be performed with cholecystokinin. In all other cases the cholecystokinin test must be carried out only as a supplement to the usual fatty meal test. After the latter has been performed, the hormone test seems indicated in the following conditions:

1. When the fatty meal test gives normal results, but the existence of a motor dysfunction of the biliary tract is clinically suspected. A normal fatty meal test and a limited or interrupted evacuation of the gallbladder or of the common bile duct after cholecystokinin are signs of alterations of the infundibulum-cystic duct junction or of the sphincter of Oddi.

2. When the response to the fatty meal is poor or negative. If the presence of extra-biliary factors (such as alterations in the stomach or duodenum) is excluded, a normal response to cholecystokinin is evidence of primary gallbladder hypotonicity; poor or no response to the hormone is, on the contrary, evidence of hypertonic stasis or of hypokinesis due to muscular deficiencies. In the first case emptying may be assisted by an inhalation of amyl nitrite.

3. When motor dysfunctions of the biliary tract have been found following gastrectomy. Cholecystokinin may then be useful in determining if the disorders are caused by alterations in the biliary tract or by an abnormal elaboration of cholecystokinetic agents.

Finally, it would appear that cholecystokinin may be of value in the therapeutic field for the continued treatment of patients with primary cholecystic atonia or with biliary stasis of the common duct due to a hypertonic condition of the sphincter of Oddi.

Preliminary results seem to support the hypothesis that cholecystokinin exerts an action also on choleresis.

Acknowledgement

The authors are grateful to Prof. JORPES for his encouragement and advice and to Vitrum AB for the generous supply of the cholecystokinin preparations.

SUMMARY

The effects of cholecystokinin on the gallbladder, common bile duct, and duodenum were investigated in 154 human subjects and 21 dogs by routine roentgenography, cinefluorography and electromanometry. The results obtained by using different doses and different methods of administration are reported, and the cholecystokinin test is compared with the traditional fatty meal test in roentgenologic examinations of the biliary tract.

ZUSAMMENFASSUNG

Die Einwirkung von Cholecystochinin auf die Gallenblase, die Gallengänge und den Zwölffingerdarm ist bei 154 Patienten und 21 Hunden mit Hilfe von rutinmässiger Röntgenuntersuchung, Kinofilm und Elektromanometrie untersucht worden. Die Resultate, die man mit Hilfe verschiedener Dosen und verschiedener Administrierungsmethoden erhielt, werden berichtet und der Cholecystochinintest wird mit der üblichen Fettmahlzeit bei röntgenologischen Untersuchungen der Gallenwege verglichen.

RÉSUMÉ

Les effets de la cholécystokinine sur la vésicule biliaire, le cholédoque et le duodénum ont été étudiés sur 154 sujets humains et sur 21 chiens par la radiographie simple, la cinéradiographie et l'électromanométrie. Les auteurs présentent les résultats obtenus avec différentes doses et différentes méthodes d'administration et comparent le test à la cholécystokinine au test traditionnel par le repas gras pour l'examen radiologique des voies biliaires.

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NORMAL VARIATIONS OF THE LEFT KIDNEY

An anatomical and radiologic study

by

J. FRIMANN-DAHL

The kidneys are parenchymatous organs situated in the retroperitoneal space close to the posterior abdominal wall at the level of the second and third lumbar vertebrae. PERNKOPF estimates the average size of the kidneys as 12 cm long, 6 cm broad and 3 cm thick, and states that there is a certain difference in shape and size, the left kidney being somewhat larger than the right. The weight varies from 150 to 200 g. The axis of the kidney is obliquely positioned in both the sagittal and frontal planes and lies parallel to the surface of the psoas muscle. Its position varies with the contraction of the psoas and also with the respiratory movements of the diaphragm.

The kidneys are described in anatomical textbooks as a pair of almost symmetrical organs. It is indicated, however, that there are some small differences between the right and the left kidney. It is well known that the left kidney lies from 1 to 10 cm higher than the right but normally this arrangement may be reversed, with the right kidney a little higher than the left. The kidney is situated more caudally on the right side than on the left, mainly because the liver intervenes and occupies most of the right hypochondrium.

The dorsal surface of the kidney is in contact with the posterior abdominal wall, lying against the psoas muscle, the diaphragm, and the quadratus lumborum muscle. On the posterior surface there are some more or less marked

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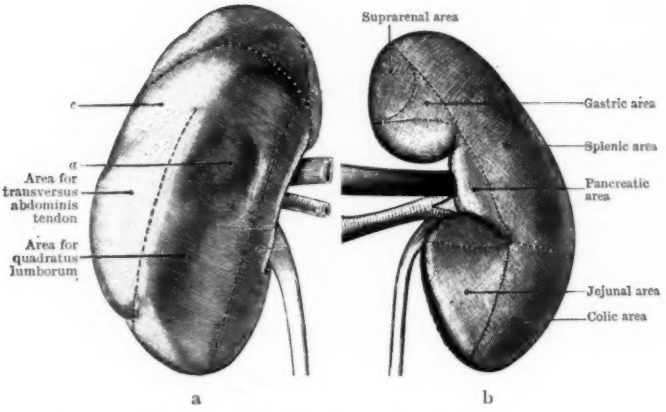


Fig. 1. a) Dorsal aspect of left kidney. Slight impressions caused by muscles and tendons upon which the kidney rests. *c* — impression of the last rib. b) Ventral surface of the left kidney showing different areas of neighbouring organs.

depressions caused by the muscles and tendons upon which the kidneys rest. When the kidney is fixed and hardened before removal, smooth elevations and ridges are occasionally observed, but usually they are not sharply defined. According to CUNNINGHAM, there is in addition to these facets on the posterior aspect often a small groove for the last rib, one for the lumbo-costal arch, and two or three minor depressions for the upper transverse processes of the lumbar vertebrae (Fig. 1a).

The ventral syntopy (Fig. 1b) is more complicated. On the right side the liver overlies the kidney anteriorly and laterally and only the posterior part is in contact with the kidney, forming the area hepatica. There is sometimes a duodenal area as well.

On the anterior surface of the left kidney three more or less defined flattened impressions are found. One of these is the splenic impression. The spleen is usually in contact with the kidney on its lateral and anterior border, but this varies largely according to the size of the spleen. Occasionally the tail of the pancreas may intervene, partly separating these organs (Fig. 2). The pancreas sometimes touches the upper pole of the kidney so that this area is slightly flattened. The stomach is only partly in contact with the kidney since the pancreas and part of the suprarenal gland intervene. Extending downwards to the lower end of the kidney is a facet for the jejunum and the colon. CUNNINGHAM further states that it is common to find the left kidney thicker and less flattened anteroposteriorly than the right, with the impressions upon its surface better marked. In this connection the fixation of the kidneys may also be mentioned.

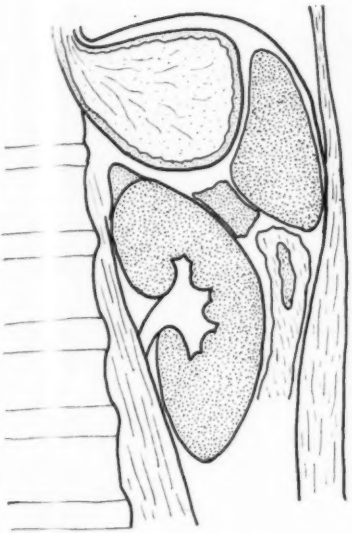


Fig. 2. Schematic drawing showing pancreas intervening between the kidney and spleen (modified after PERNKOPF).

The fact that a floating kidney is more rarely encountered on the left than on the right side is probably associated with this feature. The fixation of the left kidney may also be explained by the fact that it is anchored by its main artery, which is shorter than the right (the vein is longer on the left side).

LÖFGREN, in his anatomical studies, showed that the kidney could be divided in three lobes corresponding to three segments; there were three calyces (and pyramids) in the upper lobe, two in the middle lobe (double pair, ventral and dorsal), and two in the lower lobe. BOIJSEN used a similar classification in his anatomical studies of the vascular pattern. The same division has been adopted by SEMB and FRIMANN-DAHL in various works on diseases of the kidneys.

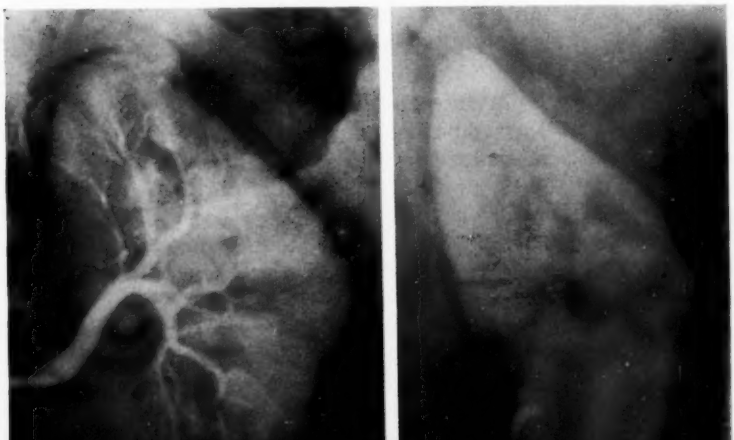
Roentgen appearances

MOËLL, in his roentgen study on the size of the kidneys, states that the kidneys are smaller in women than in men, and that in both sexes the left is somewhat larger than the right. In men he found the average dimensions to be 12.7 cm × 6.3 cm for the right kidney and 13.2 cm × 6.4 cm for the left, the total area being 129.6 cm^2 and the area per sq meter of the body surface 70.1 cm^2. The corresponding figures for women are 12.4 cm × 5.9 cm, 12.8 cm × 6.1 cm, and 119.3 cm^2, and 72.2 cm^2, respectively.

In conventional pyelography only the pelvis and the calyces are properly demonstrated. The parenchyma and the outer contour are better shown by urography, and particularly if the ureter is blocked as for instance in cases of ureteric stones. Due to accumulation of the contrast medium a so-called nephrographic effect is obtained and this provides an opportunity to determine the outer contour and shape of the kidney. By aortography a similar 'loading' of the parenchyma may be observed and the spleen is also rendered opaque (Fig. 3). Selective renal angiography produces more marked density (Fig. 4a) and the size and shape of the kidneys may be studied accurately; oblique views may prove of value (Fig. 4b). This procedure has in turn stimulated the study of the vascular anatomy of the kidneys, including the cortical pattern.

The present study is based on a series of 250 selective renal angiographies and an estimated 5 950 urographies carried out over the last three years.

Fig. 3. Urography in the course of aortography. Contrast medium in the kidneys and spleen.



a b

Fig. 4. a) Selective renal angiography showing bulging of the lateral contour. b) Latera view of kidney.

Fig. 5. Marked 'bump' in a relatively large kidney and small spleen.

Fig. 6. Bulging of lateral contour in a relatively small kidney and a large spleen.

The study indicates that in about 10 per cent of cases the left kidney has a more or less marked 'bulging' which may vary in position along the lateral border. It is usually most conspicuous in the middle segment and when marked gives the kidney an almost triangular form (Fig. 5).

The bulge is most evident in adults and in the normal-sized kidney, but even in small children a tendency to a similar deformity may be observed. In practically all cases where this deformity was present the spleen was lying closely adjacent with its renal facies parallel to the lateral border of the kidney. The lower pole of the spleen was placed just above and lateral to the elevation or 'bump' (Fig. 6). The obvious explanation therefore seems to be that the kidney in these cases 'has to carry the spleen on its back' and consequently an indentation is formed, so marked that a tumor-like elevation occurs. If unrecognized, this deformity may easily be mistaken for a cyst or even a malignant tumor. In cases of unexplained hematuria and a coincidental 'bulging' of the left

kidney, for example, misinterpretation may easily be made (Fig. 7).

Fig. 7. Bulging of the left kidney. Differentiation from a cyst or tumor is important.

Another important feature is that the kidney pelvis also shows a 'normal' anatomical variation which corresponds to the deformity of the lateral contour. The pelvis may be only slightly influenced but the calyces are often markedly changed. Different patterns observed in this material are presented in Fig. 8. As shown by the schematic drawings, it is mostly the calyces in the middle segment which are influenced. When the spleen is lying along the border, one or two calyces of the middle lobe are shortened and the kidney elongated. The lower middle calyx, which corresponds to the 'bulging', however, is elongated and larger than normal (Fig. 9). Occasionally the calyces of the upper and lower segments are also affected, being either compressed or elongated (Fig. 10). If the left kidney presents marked lobulation the bulging may be particularly accentuated (Fig. 11).

Amongst other possible causes of this deformity, the position of the pancreas must be taken into consideration. As illustrated earlier in the drawing in Fig. 2, the tail of the pancreas may intervene between the spleen and the left kidney. However, if the 'bump' could be explained as a pancreatic impression, one would expect an enlarged space between the spleen and the kidney in these cases. This has not been observed; on the contrary, the organs tend to lie closely adjacent. A third possibility is an impression caused by the 12th rib. As shown in Fig. 1a, a depression corresponding to the 12th rib is present but visible only on the posterior surface of the kidney and can therefore hardly interfere with the lateral contour. The roentgen findings have been scrutinized with this point in view and in many cases the 12th rib has been found to lie a considerable distance from the bulging or has been poorly developed. It therefore seems reasonable that this explanation may also be ruled out. The fact that the calyces are influenced by the deformity seems to prove that it must have developed early, probably at a fetal stage. A concise morphologic explanation cannot be offered in this study.

Whether the deformity of the left kidney is caused only by 'normal' spleens is a question difficult to answer and so is the question of the nature of the relationship in very small or enlarged spleens. Further experience is required. However, there is certainly a wide range of variation, for instance in cases in

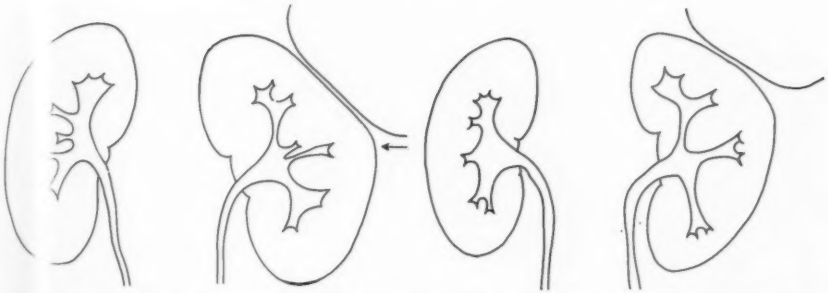


Fig. 8. Diagrammatic representations of typical normal variations of the calyces of the middle lobe of the left kidney.

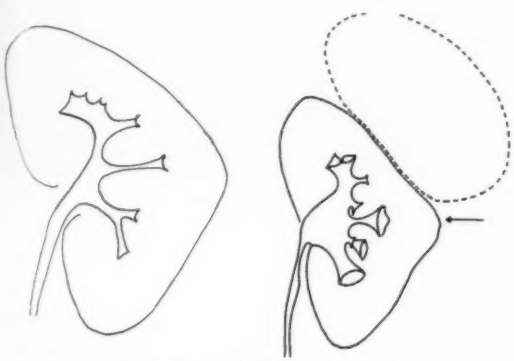


Fig. 9. Drawings showing influence of the spleen upon the renal pelvis.

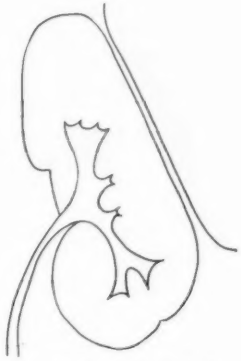


Fig. 10. Normal variation of the upper, middle and lower lobe calyces.

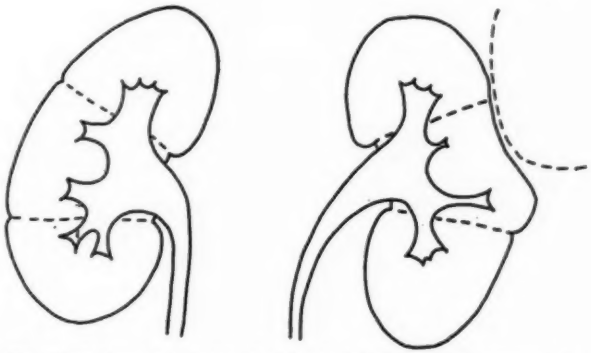


Fig. 11. Schematic drawing showing bulging of lateral contour of a lobulated left kidney.

Fig. 12. Enlarged upper pole of left kidney in combination with a small spleen.

which the spleen is nearly invisible and the left kidney lies close to the cupola of the diaphragm. The upper pole of the kidney may be correspondingly enlarged, having obviously encroached upon the space of the spleen. In such cases, the left kidney is much larger than the right (Fig. 12).

In other cases an enlarged spleen may have displaced the left kidney downwards and the deformity of the kidney which existed before the enlargement of the spleen is unchanged. Under these circumstances the left kidney is apt to be smaller than the right.

The fact that the spleen increases in size following a large intake of water has also been considered. It could have been that the deformity of the kidney was influenced secondarily by excessive fluid. We have investigated this possibility in some cases but so far no convincing results have been obtained.

Changes in the vascular pattern are, as a rule, not very conspicuous in relatively small bulgings; in other cases the vessels may be somewhat elongated or stretched in the area of the deformity (cf. Fig. 8). When there is a marked depression mainly of the upper lobe the vessels are shortened and tortuous (Fig. 13).

Finally, one may ask if a similar bulging occurs on the right side. It would not be surprising if the large overlying liver caused a deformity of the right kidney but evidently the liver lies more anteriorly than the spleen and does not

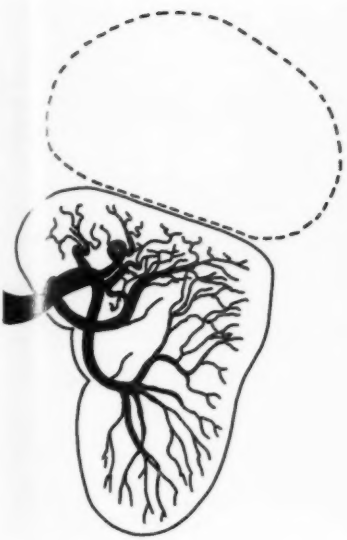


Fig. 13. Diagrammatic representation of the vascular pattern in a kidney with a marked splenic impression.

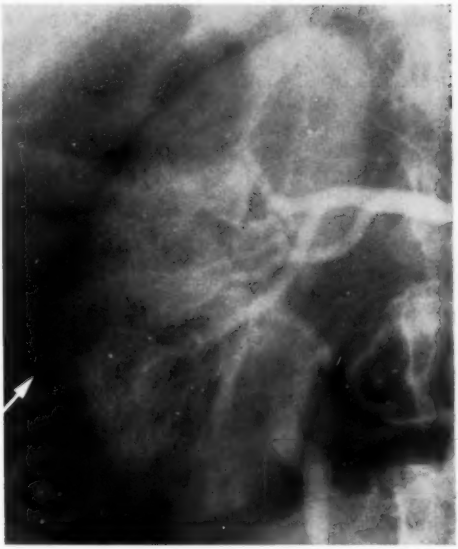


Fig. 14. Bulging of lateral contour of right kidney (one of the few observed in the period of the investigation).

influence the right kidney in the same way. In this series only one case of marked 'bulging' of the right kidney was observed, which to some extent was comparable to the findings on the left side (Fig. 14).

As far as the author is aware these observations on the appearances of the lateral contour of the left kidney and, in many cases, corresponding deformity of the pelvis and calyces and changes in the vascular anatomy, have not previously been described.

SUMMARY

A peculiar deformity of the left kidney occurring in about 10 per cent of all cases is described. This consists in a bulging of the lateral contour of the kidney with corresponding changes in the calyces; in some cases the vascular pattern is also influenced. The deformity is probably secondary to an impression made by an overriding spleen.

ZUSAMMENFASSUNG

Eine eigentümliche Deformierung der linken Niere, wie man sie in ca. 10 % aller Fälle sieht, wird beschrieben. Es handelt sich um eine Ausbuchtung der lateralen Nierenkontur mit entsprechenden Kalyxveränderungen. In einigen Fällen besteht auch eine Beeinflussung des Gefässnetzes. Die Deformierung ist wahrscheinlich Folgeerscheinung einer Impression, die durch eine übergreifende Milz verursacht wird.

RÉSUMÉ

L'auteur décrit une déformation particulière du rein gauche qui existe dans environ 10 pour cent des cas. Elle consiste en une saillie du contour latéral du rein, avec des modifications correspondantes des calices; dans certains cas, la disposition des vaisseaux est, elle aussi, modifiée. Cette déformation est probablement secondaire à une empreinte splénique.

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INFLUENCE OF SOME CHOLECYSTOGRAPHIC MEDIA ON THE SIZE OF THE GALLBLADDER

by

LARS ANDRÉN and GEORG THEANDER

Much work in recent years has been aimed at comparing the diagnostic value of various cholecystographic media, but no attention seems to have been paid to the influence of such media on the size of the gallbladder. We have found that cholecystographic media differ characteristically in this respect, and some observations are briefly reported and discussed below.

Iodoalphionic acid (Bilitrast), iopanoic acid (Telepaque) and its sodium salt (Bilijodon-Natrium, below abbreviated to Bi-Na), Biligrafin and Biligrafin-forte, have been used at the same time or alternately for routine cholecystography in our department for the last 8 years.

The number of examinations performed with these media exceeds 20 000. We received the general impression that the gallbladders demonstrated with Bilitrast, Biligrafin or Biligrafin-forte were on the average larger than those examined with any of the other media mentioned. To check this impression we compared films of a total of 30 cases examined on different occasions with different contrast media. This group included no cholecystograms showing so-called 'eccentric contractions' of the gallbladder, since this phenomenon is accompanied by a diminution in the size of the gallbladder (ANDRÉN & THEANDER 1958). The interval between the examinations thus compared varied from a few days to several months.

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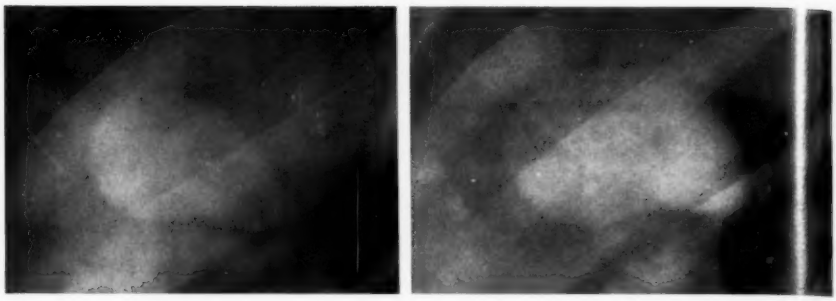

a b

Fig. 1. Cholecystography with: a) Bilijodon-Natrium and b) Bilitras[t].

Results

The differences in size of the gallbladder were often striking (Fig. 1), and the apparent effect of various contrast media seemed to support the original observation (see Table). On the other hand, comparison of the sizes of individual gallbladders on repeated examinations with one and the same contrast medium showed but slight differences and then only occasionally (Table). Furthermore, 27 cases in which the gallbladder had been demonstrated with Bi-Na were re-examined after a week with Bilitrast. No 'eccentric contractions' were present on either occasion. In 24 of the 27 cases, the gallbladder was definitely larger when demonstrated with Bilitrast. In the remaining 3 cases, no definite size difference occurred.

The combined effect of different contrast media was first studied in a few cases after simultaneous administration of Bilitrast and Bi-Na. In these the resultant size of the gallbladder was found to correspond to that obtained with Bilitrast alone. In further experiments Biligrafin-forte was administered instead of Bilitrast, since the filling process can be watched roentgenographically with

Table

Comparative size of gallbladder related to contrast medium used on repeated cholecystographies
+ larger than −

Case No.	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30
Biligrafin (or Biligrafin-forte)	+	+	=	=	=	=	=	+	+	+	+										=	=	=	±						
Bilitrast	−	−	=	=	=	=						+	+	+	+	+	+	+	+	+	=	=	=							
Bi-Na (or Telepaque).....								−	−	−	−	−	−	−	−	−	−	−	−	−					=	=	=	=	=	±

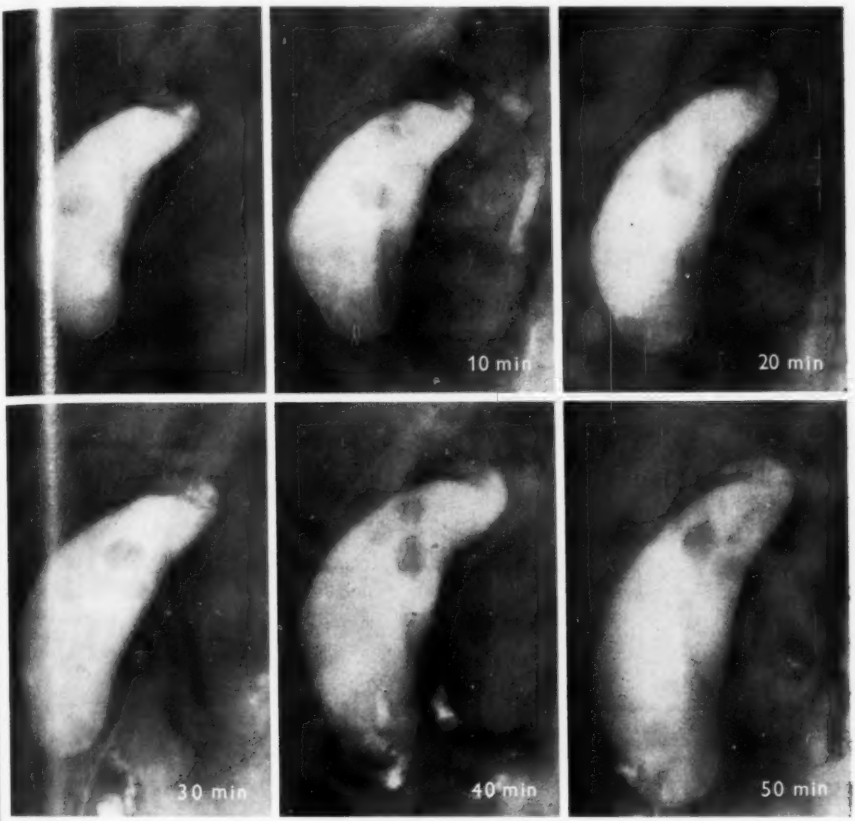

Fig. 2. Size of gallbladder on cholecystography with Eilijodon-Natrium (top, extreme left), and the gradual increase at respectively 10, 20, 30, 40, and 50 min after additional administration of Biligrafin-forte.

the Biligrafin media. When Biligrafin alone is used and the gallbladder initially contracted — such as after a fatty meal — filling is sometimes obtained of the gallbladder in the more or less contracted state after which it dilates in association with further inflow of contrast medium (THEANDER 1956). Eight patients whose gallbladders were demonstrated with Bi-Na received 20 ml Biligrafin-forte, and films were subsequently taken at 10 minute intervals. In 7 of them, the size of the gallbladder was then seen to increase gradually from that obtained with Bi-Na alone (Fig. 2).

Discussion

The observations made in the present study show that the size of the cholecystographically demonstrated gallbladder varies largely with the contrast medium used. It is not known whether any contrast medium is inert in this respect but some media apparently differ widely. This should be born in mind when roentgenographically assessing the normal range of variation of the size of the gallbladder.

The combined effect of various contrast media, differing in their influence on the size of the gallbladder, was apparently the same as when only the medium producing the larger size had been given. This seems to indicate that the effect under discussion is in reality a relaxation. Work in progress on 'eccentric contractions' also suggests the existence of some functional mechanism causing active relaxation of the gallbladder. Nevertheless, the mode of action by which different contrast media produce a different size of the gallbladder is far from properly understood. The physiologic regulation of the size of the gallbladder apparently includes the state of muscular contraction, the supply of fluid via the cystic duct, and the absorptive and secretory functions of the gallbladder mucosa, but current physiologic concepts are unable to explain how these factors cooperate to maintain a suitable gallbladder size. A new approach to this problem might be expected from further roentgenographic and pharmacologic studies of the action of cholecystographic media.

SUMMARY

The gallbladder was observed to be larger when demonstrated with Bilitrast, Biligrafin or Biligrafin-forte than with iopanoic acid or its sodium salt. When various media that differed in this respect were given in combination, the larger gallbladder size was obtained.

ZUSAMMENFASSUNG

Es wurde beobachtet, dass die Gallenblase grösser war, wenn sie mit Bilitrast, Biligrafin oder Biligrafin-forte dargestellt wurde als mit Iopansäure oder dessen Natriumsalz. Bei kombinierter Anwendung der verschiedenen, unterschiedlich wirkenden Substanzen erhielt man eine grössere Gallenblase.

RÉSUMÉ

Les auteurs ont constaté que la vésicule biliaire est plus volumineuse quand elle est opacifiée par le Bilitrast, le Biligrafin ou le Biligrafin Forte que par l'acide iopanoïque ou son sel sodique. Quand on administre en association ces moyens de contraste, dont l'effet sur le volume vésiculaire est différent, la vésicule a son plus grand volume.

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FRO THE ROENTGENDIAGNOSTIC DEPARTMENT (DIRECTOR: PROF. SÖLVE WELIN)
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UPPER TALAR ENARTHROSIS

by

FOLKE BRAHME

Upper talar enarthrosis seems to be an appropriate name for a condition in which the head of the talus is semiglobular and lies in a socket formed by the lower end of the tibia and the fibula, or the tibia alone. The literature contains only 5 cases of this anomaly (LAMB 1958). One further case has been followed since early childhood in our department and will now be reported in some detail.

Case report

A man, aged 29, with congenital shortening of the right leg, combined with genu valgum, for which surgical correction (osteotomies on the right femur and tibia) had been attempted twice in early childhood with limited success. Roentgen examination when the patient was 3 days old (Fig. 1) showed a broad bowed tibia, a short fibula and apparently normal arrangement of the tarsal epiphyses. A further examination when the patient was 3 years old disclosed a plump talar epiphysis but no signs of a separate navicular epiphysis.

He has been complaining of pain in the lower legs and feet and was recently referred for roentgen examination. Films obtained of the right and left ankles revealed that the normal ginglymoid joint had been replaced by upper talar enarthrosis. The articular surface of the head of the talus was semiglobular and lay in a socket formed on the right side by the tibia and fibula and on the left by the tibia. The right tibia was slightly bowed. There was no shortening of the fibula, but the head of the bone was deformed. Bony ankylosis was present between the talus and navicular, as well as between the bases of the fourth and fifth metatarsal bones. The arches of the right foot were flattened out, the axes of the talus and calcaneum lying almost parallel. The phalanges of the third toe were short and deformed and hallux valgus was present. There was no abnormality of the upper extremities.

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a b

Fig. 1. Right leg and foot. a) 3 days old. b) 3 years old.

The bilateral upper talar enarthrosis was the most uncommon anomaly in this case. In two of LAMB's cases the anomaly was bilateral, and in 4 it was combined with tarsal ankylosis; shortening of the lower limb was present in all the cases.

Most of the other anomalies observed in our case are fairly common. Talo-navicular fusion, however, is one of the less usual varieties of fusion in the tarsus (O'RAHILLY 1953). The first definite case of bilateral talo-navicular fusion was reported by ANDERSON in 1894 and a second case was not described until 1943 (O'DONOGHUE). Several other cases have, however, been published since then (BOYD 1944, WEITZNER 1946, CHAMBERS 1950, and others). It is widely accepted on the basis of embryologic studies that fusion in the carpus and tarsus is due to defective separation of the condensation centres in the mesenchyma. Though a fusion of tarsal bones almost always accompanies upper talar enarthrosis, there appears to be no causal relationship between the two conditions, as such fusion most frequently occurs in the absence of talar enarthrosis. One of LAMB's cases had no fusion of tarsal bones. A functional relationship between the two conditions does however exist. Any bony union between elements of the proximal tarsal bones will impair inversion and eversion of the foot. The talus, calcaneum and navicular bones form a functional unit which permits about 20 degrees of eversion and inversion of the foot. Owing to the arrangement of the muscles, movements

Fig. 2. Right ankle, a. p. Enarthrosis.

Fig. 3. Right foot. Fusions of tarsal and metatarsal bones.

in the joints are always combined and any tarsal fusion will severely impair the mobility of the foot; the limitation may be compensated in the presence of upper talar enarthrosis but only at the expense of the stability of the ankle.

The structure of the foot bones of vertebrates varies widely with numerous variants of the primate primitive plane: tibial, intermediate and phalangeal. The number of tarsals and phalanges also varies. Reptiles including the

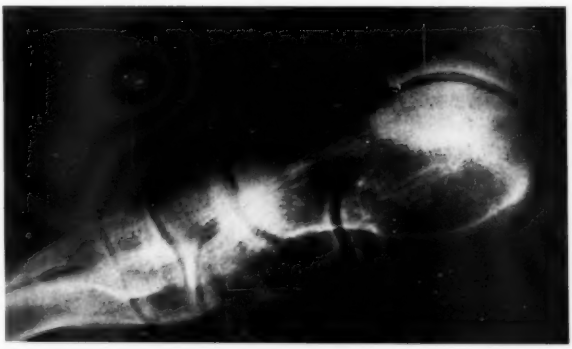

Fig. 4. Right ankle and foot, lateral.

archisaurus have only two tarsal bones, the calcaneum and the talus. A similar arrangement exists in mammals, the talus then always forming a hinge joint; a joint of the ball and socket type is never encountered. Enarthrosis cannot therefore be regarded as a sign of regression towards an earlier type of ankle joint.

Acknowledgement

The author wishes to express his thanks to Y. Löwegren and T. Askaner for the zoologic data.

SUMMARY

A case of bilateral upper talar enarthrosis, a rare anomaly of the ankle joint, is reported.

ZUSAMMENFASSUNG

Es wird über einen Fall von bilateral oberen Talus-Enarthrosis, eine seltene Anomalie des Fussgelenkes, berichtet.

RÉSUMÉ

Présentation d'un cas d'énarthrose astragalienne supérieure bilatérale, anomalie rare de l'articulation de la cheville.

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QUALITY AND CHOICE OF POTTER BUCKY GRIDS
PARTS IV AND V

by

W. HONDIUS BOLDINGH

IV. Focus-grid distance limits

The preceding parts of this series of articles were published in 1959 (BONENKAMP & HONDIUS BOLDINGH) under the same main title. Excepting the main title, the terminology and symbols in the present paper are those which will be recommended by ICRU IV.4 of which the writer is a member.

It is important that the user of a grid of given ratio and focal distance should know the extreme focus-grid distances between which it may be employed with reasonable results. The author in 1958 gave a graphical method for determining the loss V of primary radiation at a distance c from the centre line of the grid when the focus-grid distance f, the lateral decentring of the focus, the focal distance f_o, and the ratio R are known. The task of the radiologist would be facilitated if he were provided with some indication as to these limits, based on the ratio and focal distance of the grid.

The most complete information (although too complicated) on each grid would be afforded by an accompanying diagram with two graphs plotting the values of $f_1 < f_o$ and of $f_2 > f_o$ as functions of the loss V of primary radiation per cm distance from the centre line. The provision of more restricted information would appear to be a better solution and the following definition is therefore presented for linear grids.

Submitted for publication 29 April 1960.

The extreme focus-grid distances, 'focus-grid distance limits', are so determined that a loss of 50 % of primary radiation is sustained at a distance of 15 cm from the centre line of the grid, i.e. at the edges of a film 30 cm in width when a simultaneous horizontal decentring of 1 cm is taken into account.

Before we adopt this definition we should determine whether at these focus-grid distance limits reasonable results will be obtained. The contrast loss which is caused at the film edge by this 50% loss of primary radiation may be calculated by means of the formula of MORGAN. (See also the more comprehensible derivation of this formula with explanation of the symbols: Diagram 5, p. 233)

We call the intensity of the primary radiation on the film I_p' (the prime indicating that the beam has passed the grid) and the scattered radiation I_s', both with the focus centred. In the case of decentring the focus up to one of the 50 % limits, I_p' and I_s' remain unchanged in the centre line of the image, and at the edges I_s' remains practically the same, i.e. $I_s'' = I_s'$, (the double prime indicating a position at the edge of the film) whilst I_p' is halved: $I_p'' = \frac{1}{2} I_p'$.

According to MORGAN, in the first case

$$C = C_p \frac{1}{1 + \frac{I_s'}{I_p'}} \text{ and in the second case } C'' = C_p \frac{1}{1 + \frac{I_s'}{I_p''}} = C_p \frac{1}{1 + \frac{2I_s'}{I_p'}}$$

where C_p is the primary contrast when $I'_s = 0$.

Thus the contrast C'' at the edges is reduced as follows

$$\frac{C''}{C} = \frac{1 + \frac{I_s'}{I_p'}}{1 + \frac{2I_s'}{I_p'}}, \text{ where } \frac{I_s'}{I_p'} \text{ relates to the centred focus position.}$$

The value of C''/C is given in Table 1 for various values of I_s'/I_p'; the relative values of the remaining total radiation I_t''/I_t' at the edges are also shown.

According to this, the contrast at the film edges remains unchanged when the grid absorbs practically all the scattered radiation ($I_s' = 0$). However, this conclusion holds good only when both I_p' and $I_p'' = \frac{1}{2} I_p'$ are in the linear part of the density curve of the film. In all cases where the scattered radiation is not completely absorbed ($I_s' \neq 0$) the contrast at the film edges is reduced due to focussing deviations. If we assume that in general the radiation behind the grid centre will not contain more than 50 % scattered radiation, so that $\frac{I_s'}{I_p'}$ is not larger than 0.5, the contrast loss $1 - \frac{C''}{C}$ will not be more than 25 %. For

Table 1

Values of C''/C and the relative total remaining radiation I''/I'_t given for various values of I'_s/I'_p

I'_s/I'_p	0	0.1	0.2	0.5	1.0	2.0	5.0	∞
% C''/C	100	92	86	75	67	60	55	50
% I''/I'_t	50	55	58	67	75	83	91	100

distances from the film centre less than 15 cm, the loss of primary radiation is proportionally less and the contrast loss is more than proportionally reduced and wider focus-grid distance limits can be applied.

All this leads to the conclusion that the '50 % limits' may be an acceptable definition, especially as it is not obligatory but gives distance values only for guidance as regards choice, comparison and use of grids.

It should be mentioned that the above considerations are based on ideal grid construction: the losses at the edges will be larger if the strips are not exactly centred or not completely flat. However, examinations of commercially available grids by means of a photocell revealed that in the case of focussing deviations the influence of these imperfections is not important.

The author in 1958 calculated the loss of primary radiation; when no horizontal decentring was present the following losses were found:

$$V_1 = R \cdot c \left(\frac{1}{f_1} - \frac{1}{f_0}\right) \text{ and } V_2 = R \cdot c \left(\frac{1}{f_0} - \frac{1}{f_2}\right)$$

$$\text{so } f_1 = \frac{f_0}{1 + \frac{f_0 V_1}{R \cdot c}} \text{ and } f_2 = \frac{f_0}{1 - \frac{f_0 V_2}{R \cdot c}}$$

where V_1 = loss of primary radiation at the lower limit f_1
V_2 = » » » » » » upper » f_2
R = grid ratio
f_0 = focal distance
c = distance from centre line of grid

When $c = 15$ cm and for both limits $V_1 = V_2 = 0.5$ we can write for these values

$$f_1 = \frac{f_0}{1 + \frac{f_0}{30R}} \text{ and } f_2 = \frac{f_0}{1 - \frac{f_0}{30R}}$$

These limits are narrowed by $\frac{1}{15}$ = about 7 % per cm simultaneous average lateral decentring of the focus. For parallel grids ($f_0 = \infty$) these formulae

Table 2

Focus-grid distance limits f_1 and f_2 in cm as functions of f_0 and R. These values may also be determined graphically by means of Diagram 2.

Focal distance f_0	Ratio *R*				
	3	6	9	12	15
60 cm	38—170 (40—170)	48— 85 (50— 90)	53— 73 (55— 75)	55— 68 (55— 70)	57— 66 (55— 65)
90 cm	48—∞ (50—200)	64—170 (65—170)	72—127 (70—130)	77—113 (75—110)	80—104 (80—100)
120 cm	55—∞ (55—200)	77—340 (75—200)	89—203 (90—200)	96—170 (100—170)	100—154 (100—150)
150 cm	60—∞ (60—200)	88—850 (90—200)	103—280 (100—200)	112—245 (110—200)	120—212 (120—200)
∞ (parallel)	90—∞ (90—200)	180—∞ (180—200)	unsuitable under 200 cm		
50 % loss of primary radiation at 15 cm from the centre line of the grid, and with 1 cm average lateral decentring of the focus					

are transformed into $f_1 = 30\,R$ and $f_2 = \infty$. The '50 % limits' f_1 and f_2 calculated from these formulae are given in Table 2 for various ratios and focal distances, whereby 1 cm lateral decentring is taken into account. Rounded-off values are shown between brackets (in multiples of 5 up to 75 cm, multiples of 10 from 80 cm upwards, 200 for values above 200 cm).

Cross grids. It is obvious that the 50 % limits of linear grids described above are not suitable for cross grids. This is illustrated by Diagram 1, where the loss distribution in a 30 × 40 cm image (12" × 16") is shown for both types of grids, each with 50 % loss at 15 cm lateral distance from the centre.

In Diagram 1, (a) and (b), lines of equal loss of primary radiation are drawn for V = 30, 40, 50 % etc., based on the fact that for each linear grid at each point of the surface the loss is proportional to the distance c from the centre line. For both types of grid the loss at A is 50 %.

At point B, however, the loss with the linear grid is zero, whereas with the cross grid it is 20/15 × 50 % = 67 %, i.e. 2/3.

At the corners, C, of the linear grid the loss is equal to that at A, namely 50 %, whereas with the cross grid the situation is more complicated: at B,

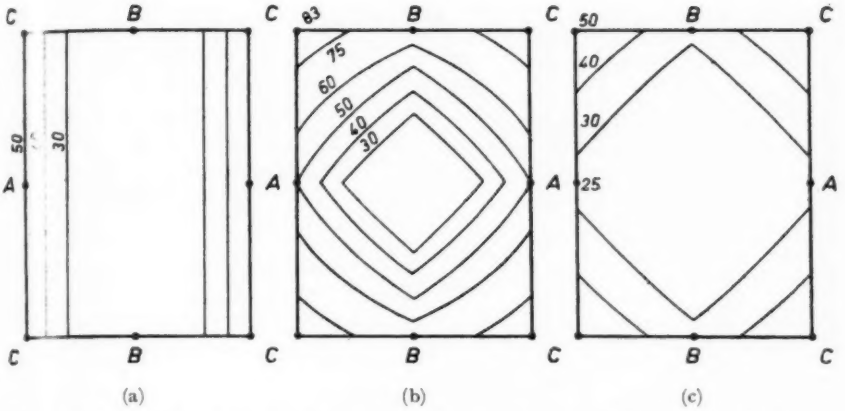


Diagram 1. Distribution of the loss of primary radiation on a 30 × 40 cm film with the tube focus at the focus-grid distance limit.

(a) Linear grid with 50 % loss in A: 0—30 % loss over 60 % of total surface
30—40 % » » 20 % » » »
40—50 % » » 20 % » » »

(b) Cross grid with 50 % loss at A: prohibitive

(c) Cross grid with 25 % loss at A: 0—30 % loss over 55 % of total surface
30—40 % » » 36 % » » »
40—50 % » » 9 % » » »

where the loss is 2/3, there remains one third of the primary intensity; at C this remaining part is halved by the other (crossing) part of the grid, so that only one sixth is left: the loss at the corners of the cross grid is then 5/6 or 83 %, which is prohibitive.

If, however, for the cross grid the loss at A were chosen as 25 % instead of 50 % the remaining part of the primary radiation would be 75 % = 3/4; the loss at B would be 20/15 × 25 % = 1/3, so that two thirds would still remain, and, at C, 3/4 × 2/3 = 1/2 would remain, i.e. a loss of only 50 % at the corners.

The loss distribution over the whole surface of 30 × 40 cm with a cross grid having a loss of 25 % at A is then slightly better than with a linear grid having a 50 % loss at A, as may be seen by comparing (a) and (c) in Diagram 1.

If we accept this '25 %' definition for cross grids, and the 50 % definition for linear grids, we can use the same formulae for calculating the focus-grid distance limits of both types, provided that for the cross grids we take into account a doubling of the ratio of one of the composing (crossing) grids.

The same holds, of course, for a graphical determination of the focus-grid distance limits. The graph in Diagram 2 is derived from the more universal one which was published earlier (Hondius Boldingh 1958 and Bonenkamp & Hon-

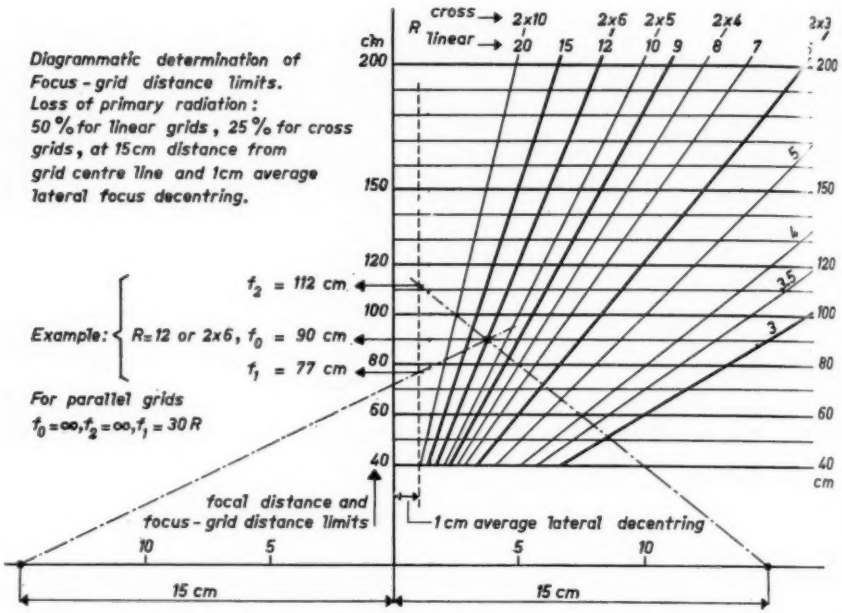

Diagrammatic determination of
Focus - grid distance limits.
Loss of primary radiation:
50 % for linear grids, 25 % for cross
grids, at 15 cm distance from
grid centre line and 1 cm average
lateral focus decentring.
Example: R=12 or 2x6, f0 = 90 cm
f2 = 112 cm
f1 = 77 cm
For parallel grids
f0 = ∞, f2 = ∞, f1 = 30 R
R
cross → 2x10 2x6 2x5 2x4 2x3
linear → 20 15 12 10 9 8 7
cm
200
150
120
100
80
60
40
focal distance and
focus - grid distance limits
1 cm average lateral decentring
10 5 5 10
15 cm 15 cm

Diagram 2. Determination of focus-grid-distance limits (50 % loss of primary radiation at 15 cm distance from grid centre line and 1 cm average lateral focus decentring). *Instructions* (for the above example): Draw —— lines through intersection of f_0 line (90 cm) with R line (12) and *read* f_1 (77 cm) and f_2 (112 cm) on intersection of —·— lines with vertical —— line.

DIUS BOLDINGH 1959, p. 249), by an incorporation of the 50 % and 25 % loss definitions for a ready determination of the focus-grid distance limits.

According to the example given in this graph, the same limits (77 and 112 cm) are found for a linear grid with a ratio of $R = 12$, as for a cross grid with $R = 2 \times 6$, both with a focal distance of 90 cm.

It may be noted that the definitions for the focus-grid distance limits given above have been adopted in the recommendations for grid terminology that are being prepared by the ICRU IV. 4 (grids) subcommittee.

V. The contrast improvement factor

The contrast caused by air and aluminium in water phantoms of various thicknesses and at various tensions have been investigated with and without a grid (BONENKAMP & HONDIUS BOLDINGH 1959, p. 153). The differences in contrast due to both types of objects have been given and it has been shown that for various grids the influence of these objects reveals itself in a similar way.

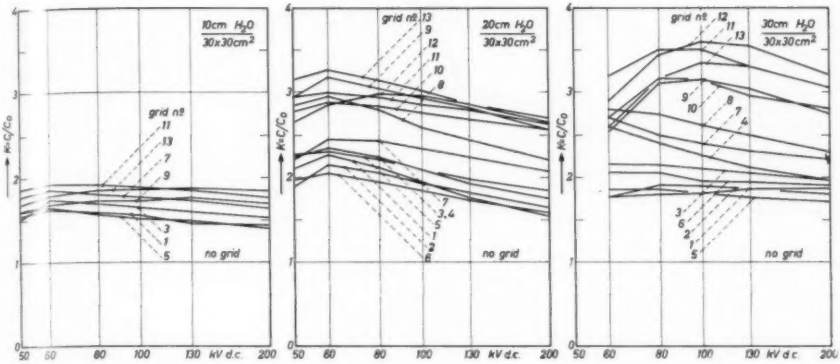


Diagram 3. Contrast improvement factors of 13 grids measured at various voltages with water phantoms of 10, 20 and 30 cm thicknesses.

One may now compare the grids by concentrating upon their contrast improving capacity, which is independent of the contrast objects. This contrast improving capacity may be evaluated by the contrast improvement factor

$$K = \frac{\text{contrast with grid}}{\text{contrast without grid}} = \frac{C}{C_o} = T_p \cdot B = T_p/T_t = \frac{\text{transmission of primary radiation}}{\text{transmission of total radiation}}$$

(See BONENKAMP & HONDIUS BOLDINGH, Part I, p. 485, and Diagram 5 in this paper.)

This factor is comparable with the definition of the selectivity of the grid (DE WAARD 1934):

$$\Sigma = T_p/T_s = \frac{\text{transmission of primary radiation}}{\text{transmission of scattered radiation}}$$

But far more than the selectivity the contrast improvement factor is directly and clearly related to what we want to know about the grid, namely the contrast improvement as compared to exposures without grid.

The contrast improvement factors of thirteen grids, measured at 50, 60, 80, 100, 130 and 200 kV with water phantoms of 10, 20 and 30 cm thicknesses, are shown in Diagram 3.

It is hardly possible in practice to discriminate between roentgenograms of the same object made with two grids having contrast improvement factors varying by only a few tenths, e.g. 2.0 and 2.2. Taking this into account the following conclusions may be drawn from the diagrams.

Table 3

Grid No.		1	2	3	4	5	6	7	8	9	10	11	12	13
Strips/cm	cm^{-1}	27	25	24	40	29	24	23	38	24	28	27	22	27
Ratio	—	3.4	2×3.1	7	11	7	6	9	15	2×7	15	15	15	15
Lead	cm^3/cm^2	0.015	0.027	0.030	0.030	0.035	0.038	0.040	0.040	0.060	0.063	0.073	0.079	0.085
content	mm	0.2	0.3	0.3	0.3	0.4	0.4	0.4	0.4	0.6	0.6	0.7	0.8	0.9
Contrast improvement factor, K_{100}		1.95	1.95	2.1	2.1	2.1	1.85	2.35	2.6	2.95	2.8	2.85	2.95	3.0

1. The curves practically do not intersect throughout the whole voltage range. This means that there are no grids particularly suitable for low voltages or others for high voltages.

2. Within practical limits the sequence of the grids as far as the contrast improvement factor is concerned is the same for various phantom thicknesses. This implies that for thinner objects the same grid as for thicker objects may be used, particularly as according to (1) it is suitable for the whole voltage range.

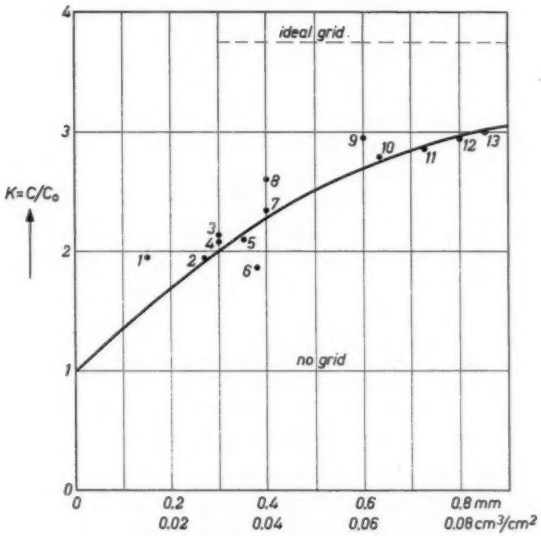
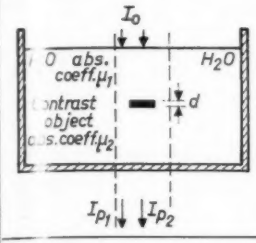
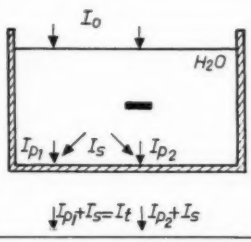
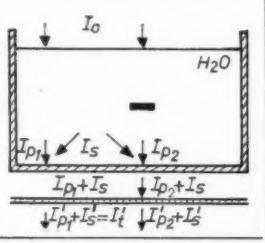


Diagram 4. Contrast improvement factor K_{100} (100 kV, 20 cm H_2O, 30 × 30 cm) as a function of the lead content.

Diagram 5. Derivation of contrast formulae

Narrow beam without grid	Wide beam without grid	Wide beam with grid
		
I_p = primary radiation	I_s = scattered radiation	I_t = total radiation
Primary contrast: $C_p=\gamma\log\frac{I_{p_1}}{I_{p_2}}=\frac{\gamma}{2.3}ln\frac{I_{p_1}}{I_{p_2}}$ $\frac{I_{p_1}}{I_{p_2}}=e^{aC_p}$ where $a=2.3/\gamma$	Contrast without grid: $C_o=\gamma\log\frac{I_{p_1}+I_s}{I_{p_2}+I_s}$ $\frac{I_{p_1}+I_s}{I_{p_2}+I_s}=e^{aC_o}$ (1)	Contrast with grid: $C=\gamma\log\frac{I'_{p_1}+I'_s}{I'_{p_2}+I'_s}$ $\frac{I'_{p_1}+I'_s}{I'_{p_2}+I'_s}=e^{aC}$
Further it is obvious that: $\frac{I_{p_1}}{I_{p_2}}=e^{d(\mu_2-\mu_1)}=e^{aC_p}$	$=\frac{I_{p_1}}{I_{p_2}}$ (2)	$=\frac{I'_{p_1}}{I'_{p_2}}$
From (1) and (2) can be derived, by eliminating I_{p_2}: which can be reduced, if aC_p and aC_o are $\langle\langle 1$ (small contrasts), to:	$1+\frac{I_s}{I_{p_1}}=\frac{1-e^{-aC_p}}{1-e^{-aC_o}}$ $\frac{C_o}{C_p}=\frac{1}{1+I_s/I_{p_1}}=\frac{I_{p_1}}{I_t}$ (3)	and correspondingly $\frac{C}{C_p}=\frac{1}{1+I'_s/I'_{p_1}}=\frac{I'_{p_1}}{I'_t}$ (4)
These are the *Morgan* formulae		
The factor $\frac{C_o}{C_p}=\frac{\text{contrast with phantom}}{\text{contrast without phantom}}=\frac{I_{p_1}}{I_t}=\frac{1}{k}$ k could be called the *contrast reduction* factor of the *phantom*		
From (3) and (4) can be derived: $\frac{C}{C_o}=\frac{\text{contrast with grid}}{\text{contrast without grid}}=\frac{I'_{p_1}}{I_{p_1}}\Big/\frac{I'_t}{I_t}=T_p/T_t=K$ where T_p = transmission of primary radiation through the grid, T_t = » » total » » » »		
K may be called the *contrast improvement* factor of the *grid*		

3. Within practical limits the shape of all the curves is the same. This means that one point of each curve, chosen at standard conditions, gives a fairly good idea of the relative quality under all working conditions. For these conditions we could choose, for example, a 20 cm standard water phantom and 100 kV d. c.

This characteristic contrast improvement factor, K_{100}, of the thirteen grids measured, with their other specific data, i.e. number of strips per centimetre and ratio, are shown in Table 3.

Diagram 4 presents these contrast improvement factors plotted against the lead content. The rough but definite relationship between the contrast improving capacity and the lead content of commercially available grids is evident. Comparing these two characteristic factors and taking into account the above mentioned difficulty of precise discrimination between grids with slightly differing contrast improvement factors the following statement may be made: the lead content is a rough but suitable indication of the contrast improving capacity of a grid, being easy to determine and independent of exposure conditions. It is therefore reasonable to refer to 'light', 'medium', and 'heavy' grids when the contrast improving capacity is considered.

The lead content may be evaluated in cm^3/cm^2, e.g. for grid No. 10 it is 0.063 cm^3/cm^2 which may be written 0.63 mm; this means that the lead content is equivalent to a lead sheet of 0.63 mm in thickness. In view of the relationship between the lead content and the contrast improving capacity being only an approximate one, the value may be rounded off to multiples of 0.1 mm and 0.063 cm^3/cm^2 read as 0.6 mm (see Table 3).

The contrast improvement factor is, on the other hand, a more precise qualitative measure but it requires standardized exposure- and measuring conditions for comparable specifications.

SUMMARY

A specification for grids of the 'focus-grid distance limits', between which reasonable results may be obtained, is described in Part IV. These limits should always be determined in the same way, according to the focal distance and the ratio. The definition of a 'contrast improvement factor' for the evaluation of the contrast improving capacity of grids is given in Part V. This factor is more accurate than the lead content recommended in Part III because it explicitly indicates the ratio of the 'detail contrast' obtained with and without a grid.

ZUSAMMENFASSUNG

Die „Fokus-Raster Abstandsgrenzen" zwischen denen man gute Resultate erhalten kann werden im Teil IV angegeben. Diese Grenzen sollten stets in gleicher Weise bestimmt werden, auf Grund einer Definition des Fokusabstandes und des Schachtverhältnisses. Die Definition eines „Kontrastverbesserungsfaktors" für die Bewertung des Kontrastverbesserungsvermögens von Rastern wird im Teil V gegeben. Dieser Faktor ist genauer als der im Teil III empfohlene Bleigehalt, da er das Verhältnis der Detail-Kontraste angibt, die man mit und ohne Raster erhält.

RÉSUMÉ

L' teur, en partie IV, donne la définition des "distances limites foyer-grille" entre lesqu iles on peut obtenir de bons résultats. Ces limites devraient être déterminées toujours de la même façon, d'après la distance focale et le rapport de la grille. En partie V il propose d'ad oter un "facteur d'amélioration du contraste" pour évaluer le pouvoir d'amélioration du cont ste des grilles. Ce facteur est plus précis que la teneur en plomb préconisée en partie III car i indique explicitement le rapport entre les contrastes de détail obtenus avec la grille et sa s grille.

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BOOK REVIEWS

ATTAINMENT AND VALUE OF PRECISION IN DEEP RADIOTHERAPY. SOME FUNDAMENTALS WITH SPECIAL REFERENCE TO MOVING BEAM THERAPY WITH 200 TO 250 kV ROENTGEN RAYS AND COBALT 60 GAMMA RADIATION. By Olov Dahl and Karl Johan Vikterlöf. 223 pages, 28 isodose charts, 90 illustrations and 25 tables. Acta radiol. (1960) Suppl. No. 189. Price Sw. Kr. 35: —.

Technical development in the last two decades has consistently offered improved possibilities for radiotherapy of deep-seated tumours. A high degree of accuracy throughout the radiotherapeutic proceedings is a prerequisite in the modern treatment of such tumours. This is necessary for a satisfactory application of current principles in each case and in every clinic. Moreover, since precision in all deep radiotherapy is essential for a detailed record of the method of treatment used for each clinical series, accuracy is essential for a satisfactory evaluation and comparison of biologic observations and treatment results between different clinical materials. The realization of treatment precision must therefore be regarded as equally important as detailed clinical observations in the development of radiotherapy.

The present publication is a continuation report on the work in progress for the attainment of precision in deep radiotherapy. As the recent work should be seen in its proper connection with the earlier investigations, a chronologic survey of the studies is first presented with a recapitulation of the basic principles and a general review of the results reported in our earlier publications. The work was concerned mainly with two forms of moving beam therapy, namely, perpendicular axial rotation therapy and perpendicular axial arc therapy.

Access to suitable anatomical phantoms was found to be necessary, and series of such models were constructed, appropriate regard being given to the anatomical and radiophysical factors involved. In the case of the thorax phantoms, both some inadvertencies in the anatomical descriptions given in text-books as well as the radiophysical properties of the functioning human lung had to be taken into consideration. As no consistent data on the radiophysical properties were available it was first found necessary to determine them experimentally. Lungs from human cadavers, inflated to a positive pressure, were employed in such a manner as to ensure that physiologically adequate conditions be attained. Radiophysical determinations of the density were carried out and, by means of corrections for divergences in blood content, a value of the average density of the functioning human lung was obtained; in further measurements a suitable lung substitute was found by means of which depth dose studies could be performed.

Since the standard isodose charts measured in homogeneous phantoms, which were presented earlier for perpendicular axial arc therapy, were intended for use in clinical practice, it was of interest to determine to what extent such dose distributions were valid in corresponding anatomical phantoms. These investigations were performed with both 200 kV roentgen rays and cobalt 60 gamma radiation and with thorax and pelvis phantoms; in these those levels were selected that were characterized by an extreme radiophysical inhomogeneity due to the presence of pulmonary or bony tissue.

Twenty-eight arc therapy dose distributions from the measurements in anatomical phantoms, judged as appropriate for direct clinical use under certain conditions, were selected. They are mainly valid for body regions of extreme radiophysical inhomogeneity, where the prognostication of the resultant dose distribution is most difficult. This means that the majority refer to the thorax region when using 200 kV roentgen rays.

A special comparison of dose distributions between 200 kV roentgen and cobalt 60 gamma irradiations with special reference to arc therapy was made. This was to evaluate further the advantages and limitations of the two radiation qualities for different clinical purposes.

Throughout the work the principle was followed that clinical measurements of dose and dose distribution should be performed as extensively as possible. As far as practicable the measurements were carried out as direct dose measurements in patients. Indirect dose measurements were also used in the form of transit dose measurements. The large measured series collected provided good possibilities for assessment of the realizability of the applied clinical measuring program, the clinical dose distributions obtained with the different methods, and the value of performing such measurements in the treatment of intra-thoracic as well as of intrapelvic tumours.

After a review of the basic procedures and the dose distribution conditions in 200 kV perpendicular axial rotation therapy of intra-thoracic lesions, a series of 303 patients with carcinoma of the oesophagus is considered; 219 of the patients were treated with the method in question. The chief reason for this was the desire to present a clinical material to which the principles of precision discussed in the volume were applied. This material was investigated in respect to some clinical, radiotherapeutic and radiophysical parameters. The treatment results are discussed for the total series under investigation, for the series of patients treated with rotation therapy and, lastly, for each of the sub-series for the upper, middle, and lower thirds of the oesophagus.

Autoreview

DIE PHLEBOGRAPHIE DER UNTEREN EXTREMITÄT. Von Robert May und Raymund Nissl. 197 Seiten und 250 Abbildungen. Georg Thieme, Stuttgart 1959. Price: DM 67.50.

Rational management of venous stasis in the legs often requires the performance of phlebography in order to establish a correct diagnosis and obtain clear indications as to adequate therapy in individual cases. As varicose veins and post-thrombotic conditions constitute the most common of all complaints in civilized countries (one-third of all women have varicose veins) their treatment cannot always be carried out at special clinics. With a view to arousing a more general interest in, and understanding for, phlebology, the authors of the book under review have endeavoured to present a clear account of the phlebographic features met with in normal and pathologic cases.

The first four chapters are devoted to normal anatomy, phlebographic technique, and problems connected with contrast media. It is refreshing to note that the authors do not seem to be ardent advocates of any particular phlebographic method; they emphasize the importance, as in all other fields of roentgen diagnosis, of aiming at exact phlebographic demonstrations of different pathologic changes in the venous system and of modifying the technique to suit the individual cases. Unfortunately, they made a departure from this rule in the case of retrograde phlebography, and left the treatment of this subject to J. D. Martinet of Paris. The chapter in which he describes his special method for retrograde phlebography is definitely inferior to the other parts of the book. Its illustrations are for the most part incorrectly interpreted and large extravasations of contrast medium in the films render a study of the conditions difficult. (In Figs 49 and 62 a, varices are said to be present in the pelvis, whereas in reality they are superficial varices in the superficial epigastric vein. Fig. 48 is stated to show the superficial circumflex iliac vein but the vessel depicted is in fact an accessory saphena magna vein with a superficial branch.) In a strikingly large number of cases the puncture was made in the great saphenous vein or some other venous branch in the groin instead of in the femoral vein; with a little practice, it is nearly always possible to puncture the latter vessel. The method described by Martinet must furthermore be considered extremely inadequate for the purpose of determining whether normal or pathologic conditions are present in the veins of the inguinal region. In a later chapter on pelvic vein phlebography

written by the authors themselves the illustrations are, on the other hand, excellent and their interpretations correct.

Twelve chapters are concerned with different abnormal conditions. The authors' own phlebograms used as illustrations in these chapters are for the most part exceedingly good, but unfortunately, on pages 129 and 130, schematic drawings of so-called valveless vein sinuses in the soleus muscle, taken from works by Cockett, have also been included; the authors still consider that such 'vein sinuses' may be present normally. Actually, these dilated, valveless veins in the calf muscles are always pathologic, and should be called muscle varices.

Pathologic communicating veins are rightly given much space. Their sites of predilection are described and their significance in primary and secondary varices is thoroughly discussed and illustrated by diagrams and instructive phlebograms. Malformations and anomalies in the venous system are treated more summarily, but are also illustrated by a few of the authors' own cases.

Injuries to deep veins caused by injection therapy are described in a special chapter. The authors over a period of 3 years watched 36 patients who had previously received injection treatment for varicose veins at other hospitals; before the treatment, these patients had been without symptoms, but afterwards their legs became swollen and post-thrombotic in appearance. The damage to the deep veins is illustrated by phlebograms and good photographs showing the clinical picture. This chapter should serve as a warning against the incautious use of injection therapy. The most common forms of varicose vein recurrence after a previous surgical intervention are also treated clearly and instructively.

The authors have devoted a special section to their own contributions to phlebography, namely experimental thrombosis (in rabbits), and a description of the so-called vein spur in the common iliac vein. This spur is, however, only illustrated by drawings, and it would hardly be unreasonable on the part of readers to demand that in a book on phlebography the so-called vein spur, given so much space in the work, should be presented phlebographically.

A list of references accompanies each chapter, but many inaccuracies and mistakes are to be found in them. On page 21, a woman investigator of the name of Birch is cited only by her Christian name, Carroll, and in a reference to the same work immediately afterwards the date is given incorrectly. On page 119, the figures 162 (1921), 71 should be placed opposite the important work by Magnus, while the data given actually refer to Lenggenhager's publication on the same page. Numerous errors of this kind occur and in a work of this class ought to have been eliminated by more careful proof-reading.

The work is an 'Ergänzungsband' to 'Fortschritte auf dem Gebiete der Röntgenstrahlen' and the typography, as is usual with publications from Thieme Verlag, is of a very high standard. Even in its present form it has a mission to fill in increasing our knowledge of phlebographic problems, but in new editions there is room for considerable improvement, especially as indicated in the present review.

Åke Gullmo

HEALTH PHYSICS. Volume 1. Progress in Nuclear Energy. Series XII. Editors: W. G. Marley and K. Z. Morgan. 602 pages. Pergamon Press, Oxford 1959. Price £ 5. 5 s.

Health physics is an important but ill-defined subject, a borderland between many natural and medical sciences. The contents of this volume are therefore varied and this, as well as the large number of authors, preclude a review in the ordinary sense. It should, however, be said from the very first that the book is most valuable as giving a comprehensive view of the state of the subject at the time of the Geneva Conference of 1958. It follows from the nature of things that the picture presented is somewhat disjointed as the contributors have mostly

work is independently, and that all parts of the subject are not equally well represented. Because of the large number of conference papers, a strict selection was necessary, but the editors seem to have made a wise choice and covered all important matters without unnecessary duplication. Some important points from papers which could not be included are given in short introductions to the various chapters.

The contents range from international and national protection recommendations, the experimental basis for decisions on maximum permissible levels, and the experiences on personnel doses in practical working conditions, to studies of water movements in deep sea trenches with a view to the advisability of using these trenches as permanent disposal places for fission products from large scale atomic energy work, a question answered in the negative. Many papers concern various dose-measuring instruments and methods, among which measurements in humans and on foodstuffs of weak activities from nuclear weapons tests fall-out and other sources, and the movements of activity in the soil-plant-animal chain command special interest. Some papers consider the hazards due to alpha emitters and methods for their safe handling. The bearing of meteorologic and geologic factors on the hazards from nuclear energy plants in normal operation and the possible consequences of a major disaster are also discussed. Regarding this, the experiences with ground disposal of fission products at Hanford and Savannah River and the extensive survey measurements after the Windscale incident are valuable.

This by no means complete survey of the contents will suffice to show the size of the circle of readers to which the present volume should appeal.

Sven Benner

RADIOLOGIC EXAMINATION OF THE SMALL INTESTINE. By Rose Golden. 2nd edit. 560 pages and 176 illustrations. Charles C. Thomas, Springfield, Ill., 1959. Price $ 28.50.

As stated in the preface to the present second edition of this work the purpose is "to continue the assembling of information which seems to be helpful in understanding the significance of the roentgen examinations of the small intestine". Since the first edition was published much new work on the small intestine has appeared and additional information concerning its anatomic structure and physiology has become available. The author's experience since 1928 has still further increased and the second edition offers the reader all the latest knowledge of value.

A general introduction, the anthropology, anatomy, and physiology of the small bowel and the normal small intestine on radiologic examination fill up the first 75 pages of the book. Conditions of developmental origin of the small intestine of the infant are then thoroughly discussed. Effects of certain drugs and food on the small intestine are also touched upon. Functional and organic disorders of the intestine including ileus and the use of the Miller-Abbott tube are treated in detail. Allergy and disorders of nutrition, inflammatory diseases, and neoplasms are some of the more important sections. The small intestine after operation, and parasitic infestation are the subjects of some other chapters. All these together with some remaining chapters on various subjects form a comprehensive and concentrated treatise on this portion of the alimentary tract.

The illustrations are almost uniformly of high quality and are well chosen.

This is a textbook of a fundamental nature, not too easy to read, but thought-provoking with its references to modern physiology, neurology and digestive chemistry. With the increasing importance of gastro-enterology this book constitutes a basis for further studies.

Ove Mattsson

DIE STREIFENATELEKTASEN DER LUNGE. Von Friedrich Heuck. 108 Seiten und 64 Abbildungen. Georg Thieme, Stuttgart 1959. Price: DM 32.

This book begins with a review of how striated atelectasis has been interpreted in roentgen films since 1928. This is followed by a chapter on general roentgen aspects and one on the mode of origin of atelectasis. The author then describes his observations in animal experiments that mainly consisted in histologic examinations of the lungs after irritation of cervical nerves at their point of exit, after irritation of the vagus nerve in the neck, and after an intrapleural acetylcholine injection. Other experiments in animals to ascertain the contractility of the unstriated muscle tissue at the periphery of the lungs are also described, and the author finally gives an account of findings in the surface of the lungs obtained with an intravital microscope. The following conclusions were drawn from these experiments: "Zur Enstehung einer Streifenatelektase ist also weder ein Bronchusverschluss noch eine Kontraktion der Lungemuskulatur, die in ihrer funktionellen Bedeutung umstritten ist, erforderlich. Wie aus meinen Untersuchungen hervorgeht, darf bei der Genese einer Streifenatelektase die vaskuläre Komponente — also die Durchblutungsstörung — gegenüber der Belüftungsstörung (Bronchusverschluss nicht unterschätzt werden".

The section dealing with the animal experiments will not be discussed in this review. On the other hand, anyone with even the slightest experience of roentgen examinations must disagree with the author's interpretation of the roentgen films of human lungs. He considered he could distinguish in the actual atelectatic area an air-conducting bronchus as an "Aufhellung innerhalb der Verschattung". These bronchi, which are marked with arrows and with accompanying sketches of the air in the brohchus, extend out almost to the surface of the lung! The bronchographic views intended to demonstrate the absence of any bronchial obstruction are not convincing.

Uno A. T. Norlin